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CONTRIBUTORS TO VOLUME II.

CABOT, RICHARD C., M.D.;
COLEMAN, THOMAS D., M.D.;
COLEMAN, WARREN, M.D.;
CUTLER, ELBRIDGE G., M.D.;
DANFORTH, I. N., M.D.;
FITZ, REGINALD H., M.D.;
GANNETT, WILLIAM WHITWORTH, M.D.;
HAYNES, IRVING S., M.D.;
LOOMIS, ALFRED LEE, M.D., LL.D.;
LOOMIS, HENRY P., M.D.;
MASON, A. LAWRENCE, M.D.;
QUIMBY, CHARLES E., M.D.;
SHATTUCK, FREDERICK C., M.D.;
SOLLY, S. EDWIN, M.D.;
TYSON, JAMES, M.D.;
WHITNEY, HERBERT B., M.D.;
WHITTAKER, JAMES T., M.D.

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SYSTEM

OF

PRACTICAL MEDICINE

BY

AMERICAN AUTHORS.

EDITED BY

ALFRED LEE LOOMIS, M. D., LL.D.,

LATE PROFESSOR OF PATHOLOGY AND PRACTICAL MEDICINE IN THE NEW YORK UNIVERSITY,

AND

WILLIAM GILMAN THOMPSON, M.D.,

PROFESSOR OF MATERIA MEDICA, THERAPEUTICS, AND CLINICAL MEDICINE IN THE NEW YORK UNIVERSITY; PHYSICIAN TO THE PRESBYTERIAN AND BELLEVUE HOSPITALS, NEW YORK.

VOLUME II.

DISEASES OF THE RESPIRATORY SYSTEM—DISEASES OF THE CIRCULATORY SYSTEM AND THE MEDIASTINUM—DISEASES OF THE BLOOD—DISEASES OF THE KIDNEYS—DISEASES OF THE BLADDER AND PROSTATE GLAND.

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CONTRIBUTORS TO VOLUME II.

RICHARD C. CABOT, M. D.,

Visiting Physician to the Channing Home, Boston, Mass.

THOMAS D. COLEMAN, A. B., M. D.,

Professor of Physiology and Pathology and Director of the Biological Laboratory in the University of Georgia; Attending Physician to the Augusta City Hospital and the Lamar Hospital, Augusta, Ga.

WARREN COLEMAN, M. D.,

Instructor of Gross Pathology in the New York University: Assistant Curator to Bellevue Hospital; Visiting Physician to the City Hospital, New York.

ELBRIDGE G. CUTLER, M. D.,

Instructor in the Theory and Practice of Medicine, Harvard Medical School; Visiting Physician to the Massachusetts General Hospital, Boston.

I. N. DANFORTH, A.M., M. D.,

Dean of, and Professor of the Principles and Practice of Medicine and of Clinical Medicine in, the Northwestern University, Woman's Medical School; Physician to the Wesley Hospital; Honorary Physician to St. Luke's Hospital; Consulting Physician to the Silver Cross Hospital, and to the Mary Thompson Hospital for Women and Children, Chicago.

REGINALD H. FITZ, M. D.,

Hersey Professor of the Theory and Practice of Physic in Harvard University; Visiting Physician to the Massachusetts General Hospital, Boston, Mass.

WILLIAM WHITWORTH GANNETT, M. D.,

Instructor in Clinical Medicine in Harvard University; Visiting Physician to the Massachusetts General Hospital, Boston, Mass.

IRVING S. HAYNES, PH. B., M. D.,

Adjunct Professor and Demonstrator of Anatomy in the New York University; Visiting Surgeon to the Harlem Hospital, New York.

ALFRED LEE LOOMIS, M. D., LL.D.,

Late Professor of Practice of Medicine in the New York University; late Visiting Physician to Bellevue Hospital, New York.

HENRY P. LOOMIS, M. D.,

Professor of Pathology in the New York University; Visiting Physician to the Bellevue Hospital, New York.

A. LAWRENCE MASON, M. D.,

Associate Professor Clinical Medicine, Harvard Medical School; Visiting Physician to the Boston City Hospital, Boston, Mass.

CHARLES E. QUIMBY, A. M., M. D.,

Clinical Professor of Medicine, New York University; Visiting Physician to the City Hospital, New York City.

FREDERICK C. SHATTUCK, A. M., M. D.,

Jackson Professor of Clinical Medicine in the Harvard Medical School; Visiting Physician to the Massachusetts General Hospital, Boston, Mass.

S. EDWIN SOLLY, M. D., M. R. C. S. (Eng.),

Consulting Physician to St. Francis Hospital, Colorado Springs, Colorado.

JAMES TYSON, A. M., M. D.,

Professor of Clinical Medicine, University of Pennsylvania and Physician to the Hospital of the University; Visiting Physician to the Philadelphia Hospital, Philadelphia.

HERBERT B. WHITNEY, M. D.,

Professor of Children's Diseases and Physical Diagnosis in Colorado State University, Denver; Visiting Physician to the Arapahoe County Hospital, Colorado.

JAMES T. WHITTAKER, M. D., LL.D.,

Professor of the Theory and Practice of Medicine, Medical College of Ohio, Cincinnati, O.

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DISEASES OF THE NOSE.

The Functions of the Nose.

The functions of the nose may be divided under four heads—(1) Respiratory; (2) Olfactory; (3) Vocal; and (4) Auditory.

(1) *The respiratory function* is the most important. Though the mouth can be used on occasion for breathing, the nasal passages are the normal and only safe route; in them the air is filtered by the coarse hairs of the vestibule, while the cilia and moistened mucous membrane catch the finer particles of dust, which later are washed off and discharged by the watery secretion. The air is warmed by passing over the network of hot bloodvessels which lie beneath the extensive surface of mucous membrane; it is moistened by the serous fluid exuded from the turbinates, which amounts daily to 500 grammes.

(2) *The olfactory function* is exercised through the medium of the surface over which the olfactory nerve is distributed. To develop the sense of smell the mucous membrane must be moist, and fine particles of the object smelled must be inhaled and thus be brought in contact with the membrane. Further than this, the mechanism of the sense of smell is obscure, but it is probable that molecular vibrations transmitted to the olfactory nerve are the initial cause, and that the pigment secreted by Bowman's glands is somehow connected with it. It aids the sense of taste.

(3) *The Vocal Function.*—The vibrations of the vocal cords set in motion by a volume of expired air striking their free edges are modified by passing behind the palate and out through the nasal fossæ, any obstruction in which causes the vocal sound to become nasal. The roof of the naso-pharynx acts as a sounding-board. Treatment of the nose or naso-pharynx will often improve the voice without being directly applied to the larynx. In all vowel sounds the air must pass through the nose as well as the mouth, which is, however, not the case with all the consonants.

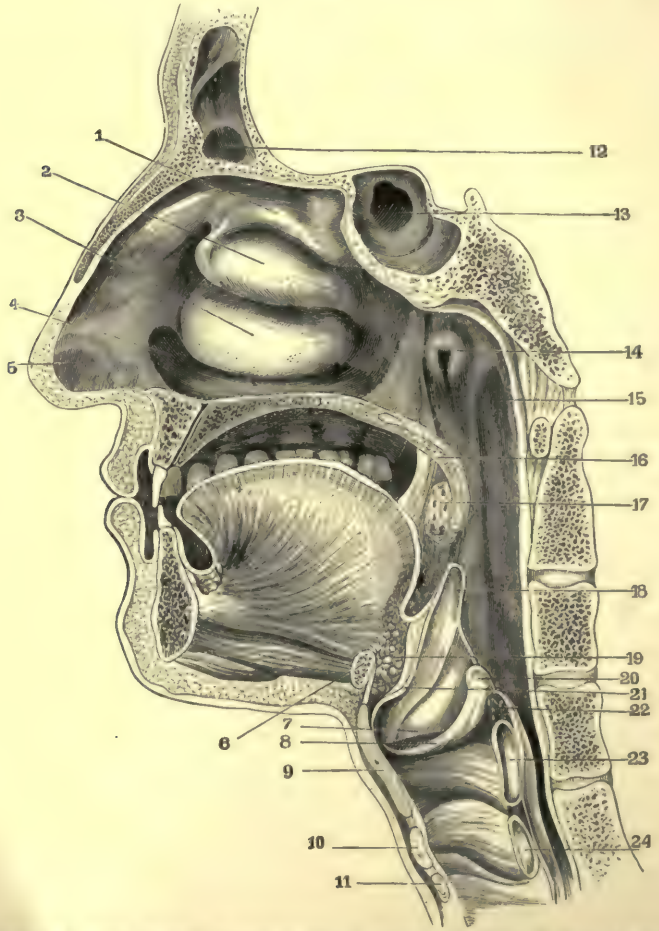
(4) *The auditory function* consists of supplying air to the middle ear through the Eustachian tubes, this being regulated by muscles

which act as valves. Obstruction of the nasal passages at once diminishes the sense of hearing.

Methods of Examination of the Nose.

A rough examination and simple treatment of the nasal cavities can be carried out by day- or candle-light, with the aid of a reflecting mirror, a nasal speculum, a small throat mirror, a spoon, and two chairs.

Fig. 1.



Vertical section of head, slightly diagrammatic: 1, superior turbinated bone; 2, middle turbinated bone; 3, lower turbinated bone; 4, floor of nasal cavity; 5, vestibule; 6, section of hyoid bone; 7, ventricular band; 8, vocal cord; 9 and 23, section of thyroid cartilage; 10 and 24, section of cricoid cartilage; 11, section of first tracheal ring; 12, frontal sinus; 13, sphenoidal cells; 14, pharyngeal opening of Eustachian tube; 15, Rosenmüller's groove; 16, velum palati; 17, tonsil; 18, epiglottis; 19, adipose tissue behind tongue; 20, arytenoid cartilage; 21, tubercle of epiglottis; 22, section of arytenoid muscle (Seiler).

To do the best work, however, the means should be somewhat more elaborate and especially adapted for the purpose. The methods vary greatly in detail, even among physicians who are equally successful in

results. As space does not permit of describing the various methods, the writer will confine himself to an outline of what he has personally found to be the preferable procedure.

The patient is seated upon a chair with a tall, straight back and a shallow seat, which should be twenty-one or twenty-two inches from the floor. The feet are placed on a footstool, with the knees close together. The chair is hooked to the floor, and away from the wall to allow of an assistant's standing behind it when required for operations. A head-rest slides up to support and fix the under surface of the occiput. The physician seats himself upon a stool with a wooden top, which screws up and down, and this seat is drawn close to the patient's knees, his own being spread out to enclose them. He is thus near enough to use his hands without leaning forward, and can keep his eyes on a level with his work, without stooping or stretching. While daylight can sometimes be used, artificial light is generally the most convenient; this should be placed near the patient's right ear. Mackenzie's light condenser attached to a ratchet-movement arm coming out from the wall, or the Toboldt light, admits of ready and varied adjustment. Gas (the Welsbach burner), electricity, or oil can be used. The room should be darkened unless the light is very powerful. To obtain a thorough examination of the parts a mirror is necessary to concentrate and reflect the light. For general purposes and use outside of the office it is best to use a mirror attached to a head-band by a ball-and-socket joint, and worn over one eye, the operator looking through a hole in the centre: by this method the physician can adjust the reflector to any movement of the patient's head, but must himself regulate the focal distance. The best head-mirror and band is that used by Bosworth. When the patient's head is easily kept steady I prefer for office use the mirror hung from a bar projecting out in front of the lamp, set at a correct focal distance.

Anterior rhinoscopy, which is nasal examination through the nostrils, requires the use of a speculum. One that is self-retaining, leaving free both of the operator's hands, is the most convenient, but of the many varieties in use most are open to one of two objections: either they depend for retention in the nostril upon a spring without a regulator, and are apt to be painful, or, if the spring is regulated by a screw, they are generally too heavy in the handle and so slip or drag out. As a self-retaining speculum an ordinary wire eye speculum is the most satisfactory. For inspection or for application, where one free hand is sufficient, a bivalve such as Bonefont's gives the best view, but when such an operation as snaring is to be done and the speculum is to be removed after the snare is in place, one with long handles, such as Ingall's, is more convenient. In using the speculum care must be taken not to give pain by inserting it beyond the lateral cartilage or pressing its point upon the septum.

When the speculum is in place, the patient's head a little depressed, and the light properly adjusted, the lower part of the nasal fossa is inspected, and a view obtained of the floor, inferior meatus, inferior turbinate, and lower part of the septum. Next, by moving the head gradually backward, the middle meatus, middle turbinate, upper portion of the septum, and superior turbinate are seen, the whole view

being completed by turning the head to either side. The inferior turbinate should appear of a pale red color, jutting out along the outer wall, with a narrow space, the inferior meatus, between it and the floor. Above it is another narrow space, the middle meatus, separating the inferior from the middle turbinate, the latter being closer to the septum and rather paler in color. Generally, only a small portion of the superior turbinate is visible. The septum should be, but rarely is, vertical. Its deviations are, however, usually unimportant unless they seriously lessen breathing space. The amount and quality of the discharge must be noted, also the color and turgescence of the membrane, after which any mucus should be removed by spraying with Dobell's solution. This should be followed by a light spray of cocaine (4 per cent.), the head being inclined forward and the spray directed so that the naso-pharynx is not unpleasantly affected by it. During the three or four minutes required for the anæsthetic effect of the cocaine the examination through the mouth can be begun. Upon again viewing the nose through the nostrils it will be found that the membrane is shrunken; if any unusual swelling remains, it is proved to be not simply congestion, but hypertrophy. The nature of the swelling, whether of the mucous or submucous membrane or of the bone or cartilage, can be further determined by gentle probing.

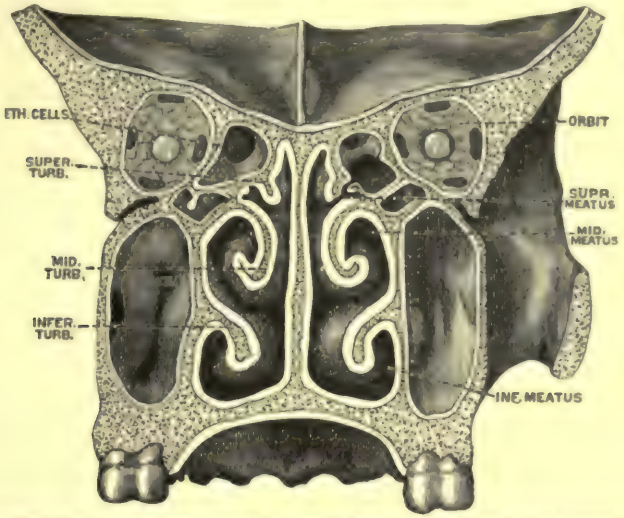
As polypi most frequently grow from the inner surface of the curled edge of the middle turbinate, its lower edge should be well shrunken with cocaine to uncover them: a pledget packed in effects this best. The shrinking of the turbinated bodies should allow of a good view of the back wall of the naso-pharynx, appearing as a glistening patch. If it can be seen that the act of swallowing causes the levator palati to contract across the lower part of the posterior nares, then the pharynx is actually visible.

Posterior Rhinoscopy.—The patient's head should be held horizontally with the mouth open: the tongue must be kept down with a tongue-depressor (Sass's), the instrument being pressed slowly, gently, and firmly forward and downward on the posterior half of the tongue, and lightly on the front half, and not inserted too far or gagging will result. To inspect the naso-pharynx the palate must be relaxed, the patient being directed to breathe through the nostrils or to say "hang," and his attention distracted from his mouth. In difficult cases, where the space is narrow, a palate hook may be used, Dr. J. A. White's being the best. A rhinoscopic mirror is then introduced on one side of the uvula between the palate and the posterior pharyngeal wall, which must not, however, be touched, as it causes gagging; an application of cocaine to it and the palate is often necessary. The mirror should be from a half to three quarters of an inch in diameter, set at an angle to the handle ranging from 105° to 130°, and the physician should either keep on hand several such instruments set at different angles or (and this I prefer) one in which the mirror may be differently inclined by pressing a lever (the adjustable rhinoscopic mirror). The handle should be held like a pen, and, when inserted, drawn to the angle of the mouth, so as not to interfere with the vision. When the mirror is placed almost horizontally, the roof and upper portion of the pharynx are seen. In adults the roof appears as a pale red, smooth, rounded dome, sometimes with

a narrow central depression, or covered with a cushion-like glandular mass, Luschka's tonsil.

Moving the mirror till it is quite horizontal reveals the egg-shaped openings of the posterior nares divided by the pale yellow septum; broad above, it narrows to its junction below with the posterior surface of the

FIG. 2.



Transverse vertical (*i. e.* coronal) section of the nasal fossæ at the plane of the second molar teeth, seen from behind (Hirschfeld).

palate. Hanging down in shadow from the upper margin of the nares are seen the yellowish colored, shelf-like superior turbinates, and at the lower margin, projecting from the side, the pale red rounded portion of the inferior turbinates appears. Between these jut out, more clearly defined and farther from the septum, the redder, rounded surfaces of the middle turbinates. Turning the mirror to one side, one may see, in profile, the funnel-shaped opening of the Eustachian tube; behind this standing out, pale and hard, from the soft pink wall is the Eustachian cushion; while behind this again, appearing in shadow, is Rosenmüller's fossa.

GENERAL DIAGNOSIS.—If, after the application of cocaine, a cushion-like thickness of the mucous membrane, somewhat yielding to the probe, remains in parts of the nasal cavity, hypertrophic rhinitis exists.

If, however, the mucous membrane is dry and shrivelled, with crusts and tenacious pus upon it, atrophic rhinitis is present.

Perhaps a drop or two of fluid pus is seen upon the lower turbinate or in the space between it and the middle turbinate; and if it, after being wiped away, returns when the patient lies down upon the opposite side with his head low, it indicates pus in the antrum. This is further confirmed if in a dark room a small electric lamp placed in the mouth reveals an opacity in the cheek of the affected side.

When blood is seen in the nose or epistaxis has occurred, the point of bleeding should be searched for. It will generally be found on the septum low down anteriorly.

The lower or middle turbinates and the septum should be lightly probed for areas of hyperæsthesia.

Semi-translucent jelly-like masses in the nose are polyps, and when they are recurrent probing frequently reveals disease of the surface or cells of the ethmoid.

If a tumor is seen, its attachment and quality must be examined with the eye and probe, and a piece put under the microscope.

Any unusual deposit of membrane, as in diphtheria, should also be subjected to the microscope.

The shape and character of any bony or cartilaginous growths, as well as deviations of the septum, require study, especially as to their effects in causing pressure or stenosis.

In observing the naso-pharynx, swelling about the Eustachian tubes must be noted, and Politzerization practised to judge of their patency, the parts being first cleaned and any discharge from them observed.

Polyps are sometimes seen protruding from the posterior nares.

Adenoid tissue or fibromatous or fibro-sarcomatous tumors may be found growing in the naso-pharynx.

When a good posterior rhinoscopic image cannot be obtained, or the view through the nostrils, after shrinking with cocaine, is not satisfactory, the physician should feel for the cushioned softness of enlarged glands, for the worm-like sensation of adenoid growths, or the firmness of a tumor by carefully hooking up the fore-finger from the mouth behind the palate. Harrison Allen has recently also advocated the use of the finger to judge of the condition of the nasal fossæ. In children the first stage of narcosis with chloroform is a necessary preliminary to these digital investigations.

Some of the principal symptoms that call for an examination of the nose and naso-pharynx are as follows:

Mouth-breathing, while occasionally only a habit, is usually indicative of nasal obstruction. It often induces a dulness of intellect and expression; while the addition of a broadened nose and a muffled voice tends to confirm the diagnosis of adenoids.

Discharge, both anteriorly and posteriorly, is frequently the complaint of the patient. A purulent or muco-purulent discharge from one nostril only is suggestive either of a foreign body or of disease of one of the accessory cavities. Spencer Watson classifies nasal discharges into "(1) liquid: (*a*) clear fluid which may be, from irritation of the trifacial or, in cases of injury, from the cerebro-spinal fluid, escaping through a fracture in the cribriform plate of the ethmoid; (*b*) turbid or mucoid fluid of simple catarrh, which may become purulent or muco-purulent—the latter generally from specific infection; (*c*) blood. (2) Semi-solid secretion indicating chronic catarrh. (3) Solid: (*a*) dry crusts from chronic rhinitis, often yellow and fetid; (*b*) diphtheritic—occasionally the diphtheritic membrane is expelled, but usually before this there is a sanious, irritating discharge from the nose, which, together with the nasal obstruction and enlargement of glands at the angle of the jaw and along the posterior border of the sterno-mastoid, points to nasal diphtheria; (*c*) rhinoliths or chalky concretions accompanied by fetid, sanious, or putrid discharge.[1]

[1] Dabney in Burnett's *System of Diseases of the Ear, Nose, and Throat*, p. 587.

Fetor is usually caused by carious bone or atrophic or purulent rhinitis.

Epistaxis, while often arising from general causes, is quite frequently indicative of intra-nasal ulceration, fibroma, or bone disease.

Sneezing, apart from that incidental to hay fever or acute catarrh, is often symptomatic of chronic rhinitis.

Many reflexes, especially asthma, are caused by nasal disease, and cough, headache, nightmare, and certain eye or ear symptoms are occasionally due to the same cause.

METHODS OF TREATMENT IN GENERAL.—*Sprays* are used for cleansing, antiseptic, astringent, or sedative purposes. Their chief and most important use is for cleansing and washing the mucous membrane preparatory to more directly curative treatment. The chief ingredients of a cleansing spray are vaporized water with an alkali, especially soda, and to this should be added an antiseptic, carbolic acid being usually the best. Dobell's solution—

(℞. Sodii bicarbonatis,	ʒj ;
Sodii biboratis,	ʒj ;
Acidi carbolici,	ʒss ;
Aquæ,	Oj.—M.)

generally fulfils the indications most satisfactorily, while Seiler's antiseptic tablets are the most portable and convenient form. There are, however, many useful modifications of these suited for special conditions. This essential cleansing by spraying can be effected fairly well with any good single-bulbed, hand-ball atomizer. In the office Sass's metal sprays attached to an air-compressor are more effective.

The astringents suitable for sprays are many ; perhaps sulphocarbolate of zinc (gr. v ad aquam ʒj) is the most generally satisfactory. Astringent sprays must at first be used well diluted and in limited quantities till the tolerance of the patient is established. Oily sprays, such as albolene, are often comforting, particularly when combined with an antiseptic sedative such as menthol, but these are not cleansing and must follow the toilet of aqueous alkaline spraying.

Of sedative sprays, cocaine produces the most immediate and positive effects, and antipyrine the most lasting. A combination of 2 per cent. cocaine and 5 to 10 per cent. antipyrine is particularly satisfactory, the cocaine alone being too evanescent, and the antipyrine at first application often producing smarting in the nose and pharynx, though not in the larynx.

Douches are occasionally more suitable than sprays in cases of atrophic rhinitis, but they are usually not as desirable because of the danger of fluid being carried up the Eustachian tubes, and because their action is confined to the lower nasal passages.

Syringing, whereby the douche can be given with greater force, has the same limited sphere of action.

Insufflations are often useful and comforting, provided the passages have first been cleansed. They should be finely pulverized, and applied in moderate quantities and evenly distributed. Ferrier's snuff or one of its modifications is particularly useful in acute coryza. Europhen

and pyoktanin are often best used in this way in certain chronic cases. Astringent powders require caution, but their action is sometimes more satisfactory than a spray, because more lasting.

In the office the compressed air used for sprays or insufflations is preferably obtained by a hydro-pneumatic pump.

After cleansing by the spray, astringency or stimulation is best effected by using the medicament upon absorbent cotton carried on a probe, triangular in shape or slightly roughened near the end so as to retain the cotton wound round it and yet admit of its easy removal afterward. Aluminum, which cannot be corroded and is easily curved, is the best material for the probe. It should have a fair-sized wooden handle.

Examples of astringents are fluid extract of ergot, full strength (gr. x–xxx ad aq. ℥j), alum, tannin (gr. x–xxx ad aq. ℥j), and nitrate of silver (gr. v–x ad aq. ℥j). A 25 per cent. aqueous solution of ichthyol, also iodine combined with glycerin in proportions suitable to the cases, are excellent stimulants.

The Probe.—The cotton-covered probe is often of service as a bougie, diminishing stenosis by pressure on the soft parts when frequently used. In the same way caustics can be applied, of which lactic, chromic, and trichloro-acetic acids, applied full strength, are the most generally valuable.

It is sometimes better to use special caustic applicators, such as Jarvis's, as the caustic is concealed till the point of application is reached, but with care they are usually unnecessary. A fresh solution of cocaine (5 to 10 per cent.) should be previously applied on cotton, and vaseline on a cotton plug afterward when there is danger of adhesions.

The galvano-cautery is less painful and more certain and definite in destroying tissue, but when furnished from a battery the latter is apt to be found out of order if its use is intermittent.

Bougies which are inserted and left to melt have an occasional use.

For epistaxis hot water (110° F.), a spray of antipyrine, 10 per cent., or of peroxide of hydrogen, an application of Lugol's solution, or the galvano-cautery to the point of bleeding is often sufficient. Severe cases may need tamponing. This is best done by the method recommended by De Roaldes,[1] which consists in passing a temporary absorbent cotton plug behind the palate, attached to a piece of thread which passes out through the mouth; the anterior nostrils are then well plugged against it with narrow strips of iodoform gauze and the posterior plug is removed.

In addition to the appliances already mentioned many surgical instruments are used, such as snares and galvano-cautery for removing morbid growths, hypertrophies, and polypi; various kinds of forceps and saws; also a surgical engine, which is preferably propelled by electricity, but if this is not obtainable water-power or a treadle can supply its place. With this engine, boring through exostoses can be best done either by trephines or burrs.

[1] *Medical Record*, Oct. 14, 1893.

Acute Rhinitis.

Definition.—Acute rhinitis is a catarrhal inflammation of the nasal mucous membrane, which is much more active and acute than the catarrh of other mucous membranes, because of the turgescence of the turbinated bodies and the excess of serous and mucous secretion.

Etiology.—Acute rhinitis is usually caused by direct exposure to cold, though it occasionally results from injury or the inhaling of irritating vapors.

Bosworth's theory that a chronic rhinitis is more often the preceding and antecedent cause, rather than the result, of repeated attacks appears reasonable, though contrary to popular opinion. He puts the matter very clearly as follows: "An exposure to cold, in the very large majority of instances, gives rise to an attack of acute rhinitis. Why this should be is easily explained: one takes cold not by exposing the whole body to a low temperature, but by exposing only a portion of the body, as from sitting in a draught, wetting the feet, etc. The primary result of this exposure is the arrest of the nutritive processes of animal heat-production in some one portion of the body; in consequence of this an additional stimulus is carried to some other portion of the economy, whereby heat-production and the ordinary nutritive processes in this latter region are abnormally increased. This latter, moreover, occurs in that portion of the system which is already weakened by some mild chronic inflammatory action. The nasal mucous membrane, with the underlying turbinate bodies, is very richly endowed with bloodvessels; moreover, the important respiratory function of the turbinated bodies involves the passage through them of a large amount of blood, which varies greatly with the hygroscopic condition of the atmosphere. Hence vascular disturbances in this region are exceedingly liable to occur—more so, probably, than in any portion of the body. Vaso-motor control, therefore, is so far weakened in this membrane that, as the result of taking cold, the inflammatory process is more apt to locate here than elsewhere. So frequent is this, indeed, that when one speaks of having taken cold an acute coryza or cold in the head is ordinarly implied. While, then, we regard an exposure to cold as the exciting cause of an acute rhinitis, the predisposing cause should always be sought for in some previously existing chronic inflammation involving the nasal mucous membrane."[1]

Pathology.—The first stage exhibits active congestion of the vessels of the turbinate bodies, with dryness of the membrane; the second, congestion accompanied by stasis and a profuse serous discharge; in the third stage this discharge becomes muco-purulent from mucus and innumerable proliferated cells which are excreted with it.

Symptoms.—The attack begins with general malaise and chilliness; next, slight fever, pains in the bones, and anorexia. The swelling of the mucous membrane causes temporary stenosis with smarting. In severe cases there is frontal headache and the eyes are similarly affected. Sneezing is usual, particularly when the wet stage is reached; with the free discharge there is a lessening of the stenosis. The discharge is acrid, often excoriating the lip. It becomes muco-purulent. Recovery is customary in about a week.

[1] Burnett's *System*, p. 611.

Complications.—The frontal sinus is sometimes involved, when a sense of fulness and distention of that region with severe frontal headache is present.

When the antrum of Highmore is invaded it is the seat of neuralgia, and toothache is experienced.

When the disease extends to the Eustachian tube and middle ear there is deafness and perhaps suppurative otitis media ; this complication, however, seldom occurs without previous chronic disease of these parts.

Diagnosis is usually easy, but occasionally acute ethmoiditis or hay fever is mistaken for acute rhinitis.

Acute ethmoiditis generally gives rise to very severe frontal headache, weakness of the eyes, and even interference with vision ; also excessive and persistent sneezing. If, however, after the passages are cleansed and cocainized and the secretion carefully wiped away with a cotton-covered probe, bright yellow pus is seen welling up from the opening of the anterior ethmoidal cells and running over the lower turbinate, the diagnosis of ethmoidal disease can be made. In this case the swelling of the cells causes the middle turbinate to be especially prominent. The persistence of the catarrhal symptoms also suggests ethmoiditis.

In vaso-motor disturbances, such as hay fever, the turbinated bodies, and not the mucous membrane alone, are usually involved. The appearances are therefore not those of acute inflammation, the membrane being of a pale bluish gray instead of a bright pink color, though in both acute rhinitis and hay fever the membrane is swollen and the secretion similar and abundant.

Prognosis.—Uncomplicated acute rhinitis is terminated in about a week without any serious symptoms, but, as Bosworth very justly says, it should nevertheless be treated, as it aggravates the underlying chronic disease, and also because it tends to develop weakness of the mucous membrane of the fauces, larynx, and parts below.

Prophylaxis.—Apart from measures pertaining to the maintenance of the general health, those who are subject to colds in the head should be very particular about their bathing and clothing.

Bathing.—The entire skin should be kept active and open by frequent cleansing with hot water and soap, but it is of the utmost importance that this should be immediately followed by cold water douching, because it tones up the cutaneous vessels and nerves and counteracts the relaxing effect of the hot water. The cleansing allows the skin to relieve the lungs and kidneys of congestion and overwork by supplementing their action of secretion. While the cold water increases this glandular action, it also enables the vaso-motor nerves to respond more rapidly and effectively to the influence of cold upon the skin, so that the vessels readily contract, shutting off or limiting the blood supply to the cutaneous capillaries, and thus preventing the blood from being chilled. When a stationary bath-tub is available and the supply of hot water liberal, I have found the most efficacious method is for the bather first to immerse himself in as hot water as he can endure, use soap freely, and then in a few minutes draw out the plug, and, turning on the cold water, sluice himself all over with it, either from the tap or shower. The water will thus be rendered lukewarm, and in this his feet should remain till he has rubbed himself dry with a rough towel.

Such a bath can be taken every morning or on alternate days, and the heat and cold regulated for the individual. The customary weekly hot tubbing, without the cold sluicing, is provocative of catarrhs, and the English morning cold tub alone is very incompletely cleansing, and too severe for many who use it, whereas the bath described gives the bather the pleasant effect of a cold bath in summer. Lithæmia, probably one of the foundations of rhinitis, is mitigated by it. Overheated rooms should be avoided.

Clothing must admit of free ventilation and not be too heavy; wool fulfils these conditions best, and should be worn both winter and summer, and very little, if at all, varied in weight, the outer garments being changed according to the season, the weather, or the heat of the house. The clothing should be equal in warmth all over the body. Mufflers and chest-protectors are abominations. The footwear is of the greatest importance. For most people woollen socks are desirable, and the outdoor shoes should cover the ankles and have stout soles, cork being inserted in them. Rubbers which are slipped off in the house are desirable in wet weather when the patient is going short distances and in and out of houses: if, however, the shoes are very stout or a brisk walk is to be taken, they may be dispensed with. Different shoes should be worn on alternate days, so as to allow of their drying out and being ventilated. Lighter shoes and socks should be worn in the evening indoors, but low slippers are risky.

TREATMENT.—After the nostrils are cleansed with a warm alkaline spray the insufflation of Ferrier's snuff is valuable; at night, Dover's powder with a hot mustard foot-bath, and three times during the day a pill of quinine, belladonna, and camphor (Rhodes's rhinitis pill), should be taken. Bosworth recommends cocainizing the nostrils, thoroughly drying with cotton, and then applying a probe tipped with chromic acid to the apex of the anterior portion of the lower turbinate.

Gérard, following Cohen, recommends chloroform inhalations till the first signs of anæsthesia appear.[1]

Bulkeley advises sodium bicarbonate, ʒj, in water, taken internally three times a day.[2] Tinctura euphrasiæ officinalis, taken internally (ʒj, ad aquam ʒss), and glycothymolin (1 to 4 per cent. in water) for a spray, used every two to three hours, are valuable remedies.

HYPERTROPHIC RHINITIS.

The correct understanding and treatment of this affection is of the utmost importance, not only because of the inflammatory and other changes it produces in the nose, but from its inducing a large number of secondary disorders of other portions of the respiratory tract, and often, indeed, of remote organs.

DEFINITION.—Hypertrophic rhinitis is a chronic inflammation of the nasal mucous membrane attended by a permanent dilatation of the bloodvessels and hypertrophy of the intervening tissues, in consequence of which the passages are narrowed, and respiration is impeded, while the lessened secretion, which is normally serous, becomes muco-purulent.

ETIOLOGY.—Probably Bosworth's opinion that deviations of the

[1] *La Médecine moderne*, Paris, 1894. [2] *Medical Record*, Jan. 18, 1896, p. 86.

septum are the most frequent cause of hypertrophic rhinitis is correct, This or any other obstruction to the equal passage of air through each nostril leads to a diminished atmospheric pressure on the bloodvessels behind the obstruction, whereby they become permanently dilated. This is the first or congestive stage, and it is followed, as a consequence of the excessive osmosis from the bloodvessels and the pressure, by fibroid and other changes in the intravascular tissue. First occurs varix of the vessels, then their destruction and ultimate obliteration; and this is known as the second or sclerotic stage. In this period enlarged turbinates and chondromatous growths appear, and changes in the epithelium, giving rise to papilloma.

When air passes through both nostrils the hypertrophy is greatest in the narrow side. When obstruction is complete the hypertrophy is well marked on the open side, being absent in the occluded nostril, in which the membrane is often bloodless.

Continued inhalation of irritating dust is occasionally a cause and frequently an aggravation. Repeated attacks of acute rhinitis sometimes induce, though they more often follow, the chronic disease. Rheumatism, gout, and lithæmia probably have no direct influence, but their diatheses undoubtedly favor the development of the nasal sclerosis. Hereditary predisposition has an influence. Congenital syphilis is a frequent cause. The use of tobacco is not a cause nor always an aggravation. Defective nutrition, especially following acute illness, and defective or irregular innervation, as in neurasthenics, are often predisposing causes.

Symptoms.—In the first stage the swelling gives rise to a feeling of obstruction in the nose, a consequent mouth-breathing, and a dry, irritable pharynx. Next come frontal headache and a hypersecretion of muco-purulent character, which flows back into the naso-pharynx, causing hawking, etc. The air which enters the respiratory tract is unmoistened because the turbinates secrete a mucoid instead of a serous fluid, and is insufficiently heated by reason of the imperfect circulation through the blood coils of the turbinated bodies. In consequence chronic pharyngitis, laryngitis, and even bronchitis, often result, also certain eye and ear affections, asthma, languor, and nervous irritability, enlarged lingual papillæ, and dyspepsia, all of which are frequent accompaniments of prolonged and severe hypertrophic rhinitis.

Inspection in the earlier stages shows the fossæ more or less occluded with soft, spongy, slightly elastic enlargements upon the lower and sometimes upon the middle turbinates. These pit on pressure with a flat probe, and shrink more or less after cocainizing. On posterior rhinoscopy similar enlargements of a dark red color are seen projecting from the lower turbinates on the nasal floor. The mucous membrane around the Eustachian orifices is generally red and swollen, and the pharyngeal tonsil is prominent.

In the later stages the turbinates are lighter in color, and the enlargements feel much denser on probing, being sometimes hard and fibrous, the anterior extremity of the middle turbinate especially so. In advanced cases these fibromatous masses often become soft and gelatinous, owing to myomatous degeneration, and appear like sessile polyps. Again, in some cases the masses shrink up. The stenosis is usually less

than in the earlier stages, except when there are special obstructions from enlargements of bone or cartilage or a diverted septum. In the naso-pharynx the membrane is thickened and fibrous and light pink or yellowish in color. The edges of the Eustachian tubes are hard and yellow. The hypertrophies are usually sessile, while those protruding from the floor of the fossa or growing from the lower turbinates or septum are generally small and corrugated, and are white or pale yellow in color. Their surface is usually rough and mammillated, the structure firm and elastic, and the distribution symmetrical.

TREATMENT.—It must be borne in mind that the disease essentially resides, not in the mucous or submucous membrane, but beneath both, in the cavernous structure, and that what has to be corrected in the first stage is the congested and varicose condition of the bloodvessels of the turbinates, which is the primary cause of the secretion of a mucoid instead of a serous fluid by the glandular structure around them. Omitting for the present the treatment of a diverted septum or other similar obstruction, which must generally be operated upon before the hypertrophic rhinitis can be permanently relieved, we will consider what is best to do to improve the turbinates themselves. There is very little doubt that in the first stages the application of a fine point of caustic to the turbinates at several spots sufficiently deep to ensure adhesion, so that the mucous membrane is pinned down to the deep structures, is the most effective method of treatment. This lessens the blood supply and astringes the vessels, so that an effusion of mucus does not thicken the normal serous exudation, and consequently the swelling of the soft parts and the stenosis which has resulted from it are also diminished. To accomplish this the simplest method, and one as effective as any, is that used by Bosworth. After wetting the end of a small probe in mucilage, it is dipped into some chromic acid crystals, and then, by heating it in a flame, a small bead of the acid is melted so that it adheres firmly to the tip. This must be freshly prepared for each application. After packing a small piece of cotton saturated with cocaine (5 to 10 per cent.) against the turbinate for four minutes, the probe is applied and held firmly on the desired spot for about twenty seconds, and then a piece of cotton smeared with vaseline is inserted. This operation is repeated three or four times, as required, at intervals of ten days.

Trichloro-acetic acid applied in the same way (as recommended by Gleitsmann) or burning with the galvano-cautery is equally satisfactory.

If, as is not unusual, there is also a subacute rhinitis, astringent or stimulating applications to the surface of the membrane, as recommended for acute rhinitis, are also needed, but all treatment must begin with a thorough cleansing of the passages.

In the later stages, when scleroma has taken place and the bloodvessels are more or less occluded and the hypertrophies permanent, the surest and most lasting benefit is by trephining if the disease is in the lower turbinate or by snaring if it is in the middle or upper.

PURULENT RHINITIS OF CHILDREN.

ETIOLOGY.—We are indebted to Bosworth for the due recognition and understanding of this important disease—important not so much on its

own account as because of its results. His views are substantially as follows: In youth the epithelial structures are especially prone to disease, the tendency disappearing in adult life and being replaced by a liability to morbid changes in connective tissue structures. During childhood the causes which give rise to inflammatory processes appear to provoke excessive development of epithelial cells. These are of two kinds—first, that whereby the new cells are added to the present structures: this is hypertrophy, of which enlarged tonsils are an example; second, that which is characterized by an activity of the mucous membrane, resulting in the throwing off of the new-formed epithelial cells by a rapid desquamation of the surface. Acute cold in the head in children is usually an acute inflammation; swelling of the glands in the vault, sometimes of the nasal mucous membrane, and very rarely of the turbinates, takes place. These attacks, especially when recurrent, are attended by an excessive muco-purulent secretion, and often result in a chronic purulent rhinitis. Commencing, as a rule, between the ages of three and five years, it runs a slow course till an age of thirteen to fifteen years is reached. The disease, which extends from the epithelium to the mucous glands, strips them of their epithelial lining, whereby they lose their power of secretion. Consequently, crusts form and the other symptoms of atrophic rhinitis develop. It is, throughout its entire course, catarrhal in character, the deep tissues being but slightly involved and ulceration and necrosis being no part of the process.

This form of rhinitis is caused, apparently, by taking cold and general hygienic neglect, or by infection in the maternal passages from leucorrhœa or gonorrhœa.[1] Scrofula and the exanthemata lead to glandular hypertrophy, and syphilis gives rise to different symptoms.

Symptoms.—The prominent symptom is a free muco-purulent discharge from both nostrils, usually bright yellow in color. There is no special stenosis, though during sleep the accumulating secretion causes mouth-breathing. There is no fœtor. Sneezing is not usual except after a fresh cold is taken.

Diagnosis.—The turbinates are somewhat swollen and of a reddish tint. There is no active turgescence, but the appearance is that of a mild subacute inflammation. On the lower and middle turbinates are seen flakes, strings, and masses of bright greenish yellow muco-pus in a semifluid state, and these are also seen on the posterior wall of the pharynx.

Except in rare cases the only other causes of a purulent discharge in children "are strumous ulceration and necrosis, and blennorrhœa, diphtheria, the presence of foreign bodies, and the late stages of acute rhinitis met with in connection with the exanthemata."[2]

With syphilis or scrofula there are marked symptoms of blood-poisoning, and the discharge is offensive, being pus mixed with blood. If a foreign body is present, the discharge is usually from one nostril. Blennorrhœa is rarely seen except in the newborn babe; it usually produces conjunctivitis; the morbid appearances of the membrane show greater activity.

[1] Tissier, *Revue mensuelle des Maladies de l'Enfance*, Paris, Jan. 27, 1894.
[2] *Diseases of the Nose and Throat*, Bosworth.

PROGNOSIS.—In the early years of the disease, with appropriate persistent treatment, cure may be effected, but in the later years the wasted condition of the epithelial and mucous glands and of the follicles makes the prognosis very unfavorable for a cure.

TREATMENT.—The discharge must be thoroughly washed out by spraying with Dobell's solution or some similar alkaline and antiseptic solution, after which the passages should be sprayed with an astringent which checks cell-proliferation, such as—

℞. Zinci sulpho-carbolatis,	gr. xx ;
Hydrargyri chloridi corrosivi,	gr. $\frac{1}{5}$;
Aquam,	ad ℥iv.—M.

Changes of astringents are sometimes advisable, and a solution of permanganate of potassium, three to five grains to the ounce, is often of benefit in controlling cell-proliferation. Watery solutions are the best, though powders, because they can be carried about and so used frequently, are often of service. Boracic acid alone, or iodol and magnesium carbonate in equal parts, make a useful powder. Cod-liver oil is often taken with benefit, and general hygienic rules must be enforced.

ATROPHIC RHINITIS.

DEFINITION.—Atrophic rhinitis is commonly known as ozæna on account of its fœtor, but as the same malodor may accompany other nasal disorders, and, as it is characterized by an atrophied or cirrhotic condition of the lining membrane, and sometimes also of the bones of the nose, the better name is atrophic rhinitis. It rarely develops before the age of fifteen or after that of twenty-five.

ETIOLOGY.—Its causes are still in dispute, the majority of rhinologists agreeing with Mackenzie that it is the last stage of hypertrophic rhinitis, while Bosworth contends with considerable force that hypertrophic rhinitis never leads to the true atrophic form (though there may be a shrinking up of some parts after long-continued hypertrophy), and that it is the outcome of the purulent rhinitis of children.

"Commencing in a desquamative inflammation of the nasal mucous membrane, there is set up the disease described in the previous chapter under the name of Purulent Rhinitis, which, as was there suggested, constitutes the early stage of the disease under consideration. We may describe atrophic rhinitis, then, as that form of catarrhal inflammation of the nasal mucous membrane which, developing in the direction of a glandular atrophy, leads eventually to a more or less complete destruction of the muciparous glands and follicles, together with a true cirrhotic condition of the mucosa proper, resulting in a condition of the nasal mucous lining in which the prominent and characteristic symptom consists in the formation of crusts and scabs, which, lodging in the sinuous passages of the nose, undergo decomposition, and become the source of a fetid and offensive odor." [1]

Mackenzie writes: "The existence of syphilis or other diathetic conditions undoubtedly predisposes to the atrophic process, and I am of

[1] *Diseases of the Nose and Throat*, p. 162.

the opinion that the constant and habitual use of alcohol is a prolific source of the new formation of connective tissue, just as it leads to similar changes in other organs, notably the liver and kidney." [1]

Elsewhere he writes: "It is indeed doubtless true that certain diathetic conditions, and especially the inherited form of syphilis, as well as certain imperfectly understood personal peculiarities, exert an important influence in the determination of the atrophic stage of rhinitis; but there is a form which, in the present state of our knowledge, is indicative of no particular dyscrasia, traceable to no particular vice of constitution, occurring in the healthy as well as in the badly nourished and cachectic, the clinical and histological history of which contains nothing to differentiate it from the atrophic stage of a simple catarrhal inflammation." [2]

Bosworth, on the other hand, writes as follows: "What has been said as to the causation of purulent rhinitis applies of course to the atrophic form of the disease. In the chapter on the former the statement was made with marked emphasis that atrophic rhinitis bore no possible relation to syphilis in any of its stages or manifestations. The same broad statement may be made in regard to tuberculosis and scrofula." [3]

Theories that the cause may be a ferment or micrococcus have been advanced, but none of them have yet been established.

My own clinical experience agrees with the view that it is not the last stage of hypertrophic rhinitis. If it were, considering the great prevalence of this disease, we would surely find many more cases of the atrophic form than we do. My colleague, Dr. P. F. Gildea, impressed by the frequent presence of ethmoiditis in cases of atrophic rhinitis, offers the reasonable theory that many cases are the result of an ethmoiditis which developed in childhood.

Symptoms.—The serous flow is lessened and the mucous is increased. The thick discharge is not easily expelled, and lodges in the crannies of the fossæ, drying into crusts in the later stages. These crusts obstruct the passage of air, and, being retained, decompose, and this is the most probable cause of the fœtor. While erosions and slight epistaxis occasionally occur, no real ulcerative process appears; the cartilaginous septum is, however, sometimes perforated, probably from the patient picking off the crusts.

Dryness of the pharynx sets in early, because the air enters through the nose without previous moistening. Inspissated mucus hangs from the vault, causing much irritation, and is only dislodged after vigorous efforts. The pharyngeal wall, as seen through the mouth, is dry and glazed. The dryness of the inspired air desiccates the mucous secretion of the larynx and bronchi, causing irritation, hawking, hoarseness, and even spasm of the glottis. Slight deafness is common, and sometimes this is serious. Bosworth reasonably ascribes it to the air entering the middle ear unmoistened and unwarmed.

Diagnosis.—Anterior rhinoscopy shows the fossæ more or less filled with peculiar greenish gray crusts, their color being characteristic. On

[1] *System of Diseases of the Ear, Nose, and Throat*, Burnett, p. 664 and p. 671.
[2] Ibid.
[3] *Diseases of the Nose and Throat*, p. 168.

removing these there is seen, apparently oozing from the fissure between the lower and middle turbinates and from beneath the upper turbinate a healthy-looking whitish yellow muco-pus. After cleansing, the membrane looks fairly normal, except that in long-standing cases it is bloodless and attenuated, and the bones are shrunken; sometimes the turbinates almost disappear, nothing remaining, in some cases, but slight ridges in place of the turbinates, and the fossæ show an unusually large cavity. The naso-pharynx and pharynx have a dry, hyperæmic appearance, with plugs of hardened mucus.

PROGNOSIS.—Atrophic rhinitis generally resists all treatment. It is doubtful if a case has ever been cured, but persistent cleansing, carried out three or four times a day, will keep the patient comfortable and mitigate the odor.

TREATMENT.—The cavities must first be sprayed out with Dobell's solution. It is essential that all the crusts be removed. To effect this, forceps or a cotton-covered probe is often needed. This must be done at the office daily till the crusting lessens, when the visits may be less frequent, but the patient must also spray his nostrils two or three times a day with a solution of permanganate of potassium gr. iij to ℥j.

The galvano-cautery lightly applied to the turbinates is often of service, as is also swabbing out with a 25 per cent. solution of ichthyol.

When erosions are present Lugol's solution, cautiously painted on them, is often valuable.

Applications of a solution of silver nitrate, ℥j to ℥j, or alcohol diluted, or a weak alcoholic tincture of galanga, sprayed behind the palate, are sometimes of service.

Braun's treatment by vibratory massage is highly endorsed.

In general treatment the common tendency to catch cold must receive due consideration.

CROUPOUS RHINITIS.

Croupous rhinitis is an acute inflammation of the mucous membrane, upon which is seen a false membrane. It is usually more severe and prolonged in children than in adults. It is attended with shivering, fever, malaise, and sneezing; the discharge, which is at first free and watery, becomes muco-purulent. More or less stenosis from swelling and the presence of the membrane exists. The membrane, which is pearly-white, generally extends to the junction of the lining membrane with the skin. Its character is recognized by its being easily detached without bleeding. It is thin in adults, but in children is apt to form a soft pultaceous mass. It is probably caused by a microbe. Sometimes there appears to be a connection between the disease and defective plumbing. It not infrequently follows burning or cutting in the nose. It usually lasts in adults about ten days, and in children about a month. It is a constitutional affection, and is most benefited by tincture ferri chloridi taken internally and also applied topically. Iodoform insufflations are also of great service.[1]

[1] Vladar, *Pester medicinish. Chirurgischer*, Budapest, Nov. 16, 1894.

Non-malignant New Growths in the Nose.

Myxomata (Polypi).—Definition.—Polypi are the most common form of neoplasm in the nose. They resemble the pulp of a grape, have a moist glistening surface, are semi-opaque, have a grayish color, and are found both singly and in groups. Bosworth says the proportion of frequency is about 1 of polypi in every 11 cases of catarrhal trouble.

Polyps may be either pedunculated or sessile, "and vary in size from that of a birdshot to the limit of the nasal cavity."[1] They are an hypertrophy of the glandular and connective tissue elements of the mucous membrane, with an epithelial envelope. They commonly grow from the middle turbinates, occasionally from the septum. These growths occur most often in males, and they rarely appear before the age of fifteen, but they may be found at any age. Their causation is obscure, though they seem to develop most often in cases which have a chronic excess of serous secretion.

Symptoms.—Violent sneezing and a profuse watery discharge are early symptoms; later, stenosis and an irritating sense of pressure are experienced. Commencing in one fossa, they usually spread to the other. The sense of smell becomes obliterated. Occasionally there is a flow of bright yellow pus, which is generally caused by disease of the antrum through obstruction of its orifice.

Reflex disturbances and aural and ocular symptoms are not infrequent. There is in advanced cases a broadened look to the nose and a dead voice. Bronchitis and laryngitis often result.

Diagnosis.—The nose should be cocainized, when the polyps will show up distinct from the mucous membrane, which appears shrunken, while they remain swollen. They indent with probing, and when protruding below the middle turbinate are found to be movable.

Prognosis.—Polyps are not of themselves dangerous to life, but become serious by reason of the obstruction they cause. They probably do not spontaneously suffer a cancerous degeneration, but this perhaps occasionally happens as the result of harsh efforts at removal.

Treatment.—The treatment is purely surgical. The important thing is to remove the polypi completely by the cold wire écraseur or snare without injury to the healthy tissues.

Polypi must again be looked for at the end of a week, as sometimes others hang down which had previously been crowded out of sight; and these inspections should be repeated once a month for four or five months.

Rhino-scleroma is a very rare disease. It is said to be less infrequent in Hungary and America than in other countries. Alvarez[2] believes it to be due to a microbe. It consists of the formation of plates of dense fibrous connective tissue either in the deep submucous membrane of the nasal cavities or in the corium of the skin of the nose or lips. It is also said to invade occasionally the pharynx, larynx, and trachea.

Rhino-scleroma can be distinguished from a syphilitic gumma, epithelioma, or keloid by its hardness, ivory-like appearance, the absence of a tendency to inflammatory or ulcerative processes, and its slow,

[1] *Diseases of the Nose and Throat*, Bosworth.
[2] *Sajous's Annual*, 1895, vol. iv. p. 13.

painless growth. It is not dangerous to life, but may cause much mechanical inconvenience. It is believed to be incurable. Various methods of treatment are described in Sajous's Annual.[1]

Fibroma.—This disease is comparatively rare; it is most frequent in males between the ages of fifteen and thirty years.

It originates in the sheaths of the vaso-motor nerves. The structure is similar to fibromata in other regions, but differs in being highly vascular. Consequently epistaxis is frequent, and during operations the bleeding is excessive.

There is rarely any pain. Fibroma usually originates in the roof, and as it grows forward it spreads the nasal bones and gives rise to the appearance called frog face. The pressure caused by its growth occasions a variety of symptoms.

After cleansing and cocainizing the nose, a smooth, glistening, pinkish-red tumor, of a resisting texture, is seen. A needle inserted will distinguish it from osteoma or chondroma. Its point of origin is important, but sometimes difficult to determine. When intra-nasal it is usually unilateral, and when pharyngeal bilateral.

The cold snare with a thick wire is the best instrument for its removal. When large, Casselberry's plan of first dividing it with the galvano-cautery knife before removal is desirable.

Papillomata.—These warty growths are not common in the nose; they are hypertrophies of the normal papillæ. Their appearance is characteristic. When they grow near the entrance of the vestibule they are small, hard, mammillated, and of a grayish pink color: the most frequent site, however, is on the inferior turbinates, where the growths appear softer, larger, pinker, and somewhat pedunculated.

Nasal papillomata occur most frequently in females and in early adult life. Their growth is slow, and gives rise to very slight symptoms unless stenosis ensues. The best method for their removal is by the use of the snare or scissors, followed by light cauterization.

Adenomata.—Pure adenomata probably never exist in the nasal cavities because of the limited amount of glandular tissue.

Cystomata are very rare. They have the appearance of a single polypus and call for similar treatment.

Angiomata are also very rare in spite of the great vascularity of the tissues. They are easily recognized by their red or purple color, and by the fact that an indentation left upon them by a probe does not quickly fade out, and generally produces hemorrhage. They are best removed by a snare—the Jarvis preferably, because its slow movement limits the hemorrhage, which is the chief danger in the operation.

Chondromata.—These cartilaginous tumors are not common. They closely resemble fibroma, but are harder and more gritty when probed, and the discharge is usually offensive. Puncturing with a needle will serve to distinguish them from osteoma by revealing a lesser degree of hardness (or bonyness).

Their removal is not attended with hemorrhage; the method should be determined by their character and size.

Osteomata.—External deformity and pain are generally the earliest symptoms. The bony character is easily recognized, though sometimes

[1] In vol. iv. D. 14.

the mass is broken off from its base, when it may be mistaken for a rhinolith.

These tumors grow from the periosteum: either they are cancellous in structure with a hard outside shell, or they consist of dense bone throughout.

When large and hard, an external operation is usually necessary to gain access to the pedicle, which, being of cancellous structure, is readily divided. Osteomata do not recur. Bony cysts are sometimes found which are expansions of the turbinates; they are readily removed by a snare.

Deformities of the Nasal Septum.

These are important only when they cause nasal obstruction and dislocation of the columnar cartilage, interfering with the free passage of air through the nostrils or producing reflex symptoms by pressing on the turbinates.

There is little doubt that nasal obstruction, which is so common a result of these deformities, is the most frequent cause of chronic hypertrophic rhinitis.

The deviations of the septum sometimes show a convexity in one nostril and a corresponding concavity in the other, and the concavity may be filled with a buttress of new bone or cartilage. Again, the deflection may be sigmoid; that is, having a horizontal or vertical curve projecting into each nostril, or spurs or ridges may grow from the septum into one or both nostrils. These are occasionally united to the turbinates, thus forming synechiæ or bridges.

Some irregularity of the septum has been found in the proportion of about three out of every four civilized white persons, while among the uncivilized races and among negroes the proportion is only about 20 per cent.

These irregularities are rare before the age of ten years.

Etiology.—It is probable that they arise from various causes, and these are subjects of interesting speculation. While it is known that traumatism is often the cause, this is probably not so frequently the case as is generally believed. The theories recently advanced by Mayo Collier[1] seem to the writer most plausible. Briefly stated, his argument is as follows: He traces the evolution of deviations of the septum thus: first, there is some temporary obstruction of one nostril; the air in passing rapidly through the open side exhausts that from the closed chamber, as in the action of a spray tube, the result being that the air pressure in the open nostril bends the septum toward the closed side.

In a study made by the writer of the relation between nasal obstruction and pulmonary phthisis[2] from an analysis of two hundred cases the evidence seemed to show that both the nasal and pulmonary disease usually began on the same side of the body, indicating a developmental deficiency as the predisposing cause. This theory falls in with Collier's as explaining why one nostril is first more readily obstructed, and then,

[1] *Journal of Laryngology, Rhinology, and Otology*, Rebman, London, March, 1896, p. 117.

[2] "The Relations of Chronic Nasal and Laryngeal Diseases to Pulmonary Tuberculosis," *Journal of the Amer. Med. Assoc.*, Sept. 25, 1894.

under air pressure, more easily collapsed. Collier further suggests as a reason why the air pressure is greater on one side of the septum than on the other that, owing to defective innervation, the muscles sometimes fail to dilate the nostrils during inspiration, and this again favors the view that congenital deficiency of one side of the body often predisposes the septum to bend toward the weaker side. The fact that Durêt found septal growths to be apparently composed of inflammatory material is not adverse to these theories. A highly arched palate is claimed as one of the indirect effects of traumatism. However, it is not possible to give further space to this interesting discussion.

SYMPTOMS.—These deviations are quite commonly accompanied by a lack of symmetry of the external nose which sometimes amounts to deformity. The initial condition is nasal obstruction with more or less interference with respiration; the second condition is a consequence—viz. chronic hypertrophic rhinitis. "As the result of stenosis, either in one or both nares, the air immediately behind the point of deflection is rarefied by each act of respiration; consequently the soft spongy membrane covering the turbinated bones is subjected to a sort of dry cupping process, as it were, by which its vessels become permanently dilated as time progresses." Thus writes Bosworth, who further goes on to explain how, as a result of the increased blood supply, there is increased growth, and therefore thickening, of the membrane. He also points out that this hypertrophy is usually greatest on the side through which the air passes most freely, and that when a nostril is completely impervious to air its membrane is bloodless and collapsed. For these reasons it is common to find hypertrophied turbinates fitting into concavities of the septum.

As results, stenosis, epistaxis, and reflex disturbances, with sneezing and watery discharges, often appear. The voice too is apt to be weakened, and ear troubles may develop from the same cause.

DIAGNOSIS is readily made by anterior rhinoscopy, these deformities rarely extending to the posterior nares. Probing will generally demonstrate the nature of the obstruction.

The question whether the stenosis is sufficient to warrant surgical interference is the important matter to decide.

If an oily vapor, sprayed into one nostril, should not issue freely from the other, the mouth being closed, nasal stenosis exists, provided there are no growths in the naso-pharynx. Also when the patient expires forcibly first through one nostril and then through the other, the mouth and one nostril being kept closed, it will be noticed that the pitch is higher through the obstructed nostril.

TREATMENT.—Electrolysis or ignipuncture[1] can be used when surgical procedure is not permissible. The various methods of straightening the septum are painful, tedious, and frequently not lasting in results.[2] They are seldom advisable except to remedy a marked facial deformity. Of the various plans, that of first punching or straightening the septum and then applying externally the nasal clamp splint[3] is perhaps the best.

[1] Moore, *Journ. Laryngol.*, London, May, 1894.
[2] On this subject see H. Levy, *Dissertation Inaugurale*, 1894, Laverne.
[3] See Jarvis, p. 57, vol. ii., Burnett's *System of Diseases of the Ear, Nose, and Throat.*

The *columnar cartilage* is occasionally dislocated so as to obstruct the nares, when it must be excised.

Foreign Bodies in the Nose.

Foreign bodies are most frequently inserted through the nostrils by children or lunatics. Common objects so introduced are buttons, fruit-stones, coins, pebbles, beans, and pieces of wood or paper.

Vomited food occasionally lodges in the posterior nares. Bullets or gun-shot may penetrate through the walls, and these, and even teeth, have occasionally been found in the nose. A. MacCoy reports a case where the tooth was found with the root upward; he attributes various reflex symptoms to these misplaced teeth.

A unilateral fetid discharge with obstruction, especially in a child, is most commonly due to the presence of a foreign body. Foreign bodies will sometimes remain, and be forgotten for years before relief is sought. They may be concealed by granulations or the swollen membrane; probing, however, generally reveals their character. In children they are usually lodged between the inferior turbinate and the septum.

For the removal of these bodies cocaine should first be used, and, if possible, extraction be made through the nostril with long thin-bladed forceps or with a bent probe passed behind it, or, if this fails, with a steel scoop, which must be used with care. There is, however, no need for the haste and excitement usually exhibited. If impacted far back, it is sometimes easier to push the object into the naso-pharynx and receive it on the tip of the index finger. Occasionally a general anæsthetic is needed, and sometimes the object has first to be bored with a galvano-cautery or Sajous's method is used.[1]

Douching is generally useless and may cause aural disease.

Rhinoliths are nasal calculi whose nuclei are generally formed by some foreign substance around which the saline constituents of the mucous secretion have hardened. They may be modified in outline by the character of the nucleus or the walls of the nasal cavity, but are usually of an elongated ovoid shape.

Rhinoliths are not uncommon. It is thought that gout and nasal catarrhs are the chief predisposing causes.

The SYMPTOMS are due to the pressure. A grating feeling is experienced on probing.

Rhinoliths usually require some crushing with strong forceps before removal, and when very hard and large the use of a lithotrite or a division of the external nose or palate is sometimes necessary to extract them.

Parasites.—Living creatures are very seldom found in the nose in temperate climates, though ascarides, centipedes, earwigs, and leeches occasionally gain an entrance. In the tropics it is quite common for flies to deposit their eggs in the nasal cavities, and for maggots to be present there. In these climates the Lucilia hominiveris is the most frequent intruder. Irritation, sneezing, sanious discharge, bleeding, œdema, and catarrhal symptoms sometimes ensue. Snuffs, syringing

[1] *Lectures on Diseases of the Throat and Nose*, p. 213.

with equal parts of chloroform and water, or the forceps may be needed for their removal.

Neuroses of Olfaction.

Parosmia is a perversion of the sense of smell, so that while the sense remains complete and the nasal organ normal, yet the individual is subject to the sense of smelling odors which are not present. This is not infrequent in insanity, hysteria, epilepsy, and syphilis, and may occur in la grippe. It appears to be usually of central origin.

Hyperosmia is a condition in which the sense of smell is exaggerated. It may be connected with local irritation, or be reflex from other organs, or be caused by a central functional disturbance, or some depression of nerve force, such as neurasthenia.

Anosmia is a deficiency or absence of the sense of smell. It is occasionally congenital, and very commonly occurs during an acute catarrh or as a consequence of stenosis, when it is often unilateral. Polyps and other tumors frequently cause it. Other causes are inhaling irritating gases, traumatism, tobacco-poisoning, syphilis, paralysis, and atrophic rhinitis.

"In testing for anosmia, substances should be used that affect only the olfactory nerve, and not acrid or pungent odors that stimulate the branches of the fifth pair."[1] Experiments have proven that the fifth pair have nothing to do with the reflex disturbances of olfaction. The connection between the sense of smell and memory and other psychic actions is interesting. Saltness, sweetness, bitterness, and acidity are recognized by the sense of taste, but the sense of smell must be added in order to realize flavors. When smell is deficient there is often an increased perception of irritants.

Besides the treatment of the causes for anosmia, Althaus recommends strychnine locally, and Bosworth, stimulation by powerful odors frequently varied. For parosmia, Grazzia advises topical applications of cocaine.

Rhinorrhœa, is, according to Bosworth, an exceedingly rare disease. He reports 18 cases in all. It only occurs in adults. It develops at all seasons of the year and at all hours of the twenty-four, yet is apt to recur at certain definite times of the day. The prominent symptom is a profuse watery discharge. In some cases the discharge is passive and painless, and in others it resembles that of hay fever, being attended with intense irritation.

In the first group, Fink, Paulosen, Bosworth, and others attribute the cause to an ablation of the function of the fifth nerve, so that its inhibitory control over serous exudation is impaired. Daal believes the spheno-palatine branch is the especial seat of the lesion. Whether there is a neuritis or some other affection of the nerve is uncertain. Four cases reported in 1896 proved to be discharges of the cerebro-spinal fluid; sugar was found in the discharge.[2]

When polypi are found with rhinorrhœa they are probably a result and not a cause.

In the second group of cases attended by irritation there is evidently

[1] J. A. White, Burnett's *System*, p. 95, vol. ii.
[2] *Journal of Laryngology*, Jan., 1897, p. 8.

no paralysis of the fifth nerve, and therefore it is probable the fault lies with the sympathetic. These cases are very similar to hay fever, and their periodicity suggests that the exciting cause is found in certain unknown atmospheric conditions. Rhinorrhœa is similar to hay fever in exhibiting an underlying neurotic habit, but dissimilar in that it is not connected with disease or the impact of pollen in the nasal cavities.

The PROGNOSIS in both groups is unfavorable.

When the cause cannot be ascertained and removed the treatment is experimental.

Reflex Neuroses.—*Hay asthma*, *bronchial asthma*, and *chorea*, which are the most important of these affections, are discussed elsewhere. There is little doubt that nasal disease may give rise to almost every kind of reflex disturbance, but at the same time, in a certain proportion of the cases in which the treatment of a nasal disorder results in disappearance of the reflex symptoms, it is probable that the success is chiefly due to the effect upon the mind, and the cure is through suggestion.

Space will not permit the discussion of the numerous nasal reflexes, but attention may be called to the fact that eye symptoms as well as cough and laryngeal disturbances frequently result from nasal disease, and that vertigo is sometimes traceable to the same cause.

SYPHILIS OF THE NOSE.

Primary and secondary syphilitic lesions are very rare in the nose, but gummatous tumors are not uncommon. They seldom appear except on the septum. When they affect the septal cartilages they are usually bilateral, and when seen on the turbinates unilateral.

The SYMPTOMS are those of obstruction, with anosmia, serous discharge, and nocturnal pains in the cheek, eye, or forehead. After a time the tumor ulcerates, the ulceration generally not spreading beyond the limits of the growth. The tumor is sessile, smooth, and usually pale in color. When probed it feels hard and elastic, but cannot be indented and is immovable. It is to be distinguished from deviation of the septum by its elasticity and by there being no corresponding depression; from septal abscess by the chronic nature and the absence of fluctuation. The nocturnal pain is diagnostic of a gumma.

Antisyphilitic treatment is called for (see Syphilis, Vol. I. p. 895), and sometimes the stenosis and pain demand a scraping out of the tumor. When the gumma has broken down antiseptic cleansing with the thorough removal of crusts is most important.

TUBERCULOSIS OF THE NOSE.

Tuberculosis is very rare in the nasal cavities. Bosworth says it is most apt to appear upon the septum in the form of clusters of small tubercular nodes, which break down into shallow, irregularly rounded ulcers, grayish pink in color, from which proceeds a slightly opalescent, viscid, grayish white secretion. There is very little tenderness or pain. The surrounding membrane is of a grayish pink, ashy color. Only one case has been reported in which there was not also tuberculosis elsewhere.

The writer successfully treated a tubercular ulcer of the right middle turbinate with curetting and lactic acid, and it has remained healed for over a year. The patient has chronic pulmonary tuberculosis. The diagnosis was confirmed by the microscope. Tuberculous ulceration of the septal cartilage occurred during the last few weeks before death in a case of intestinal tuberculosis. These were the only two cases the writer has seen in which the diagnosis was certain.

Lupus of the Nose.

This disease very rarely invades the nasal cavities. When present there is more or less stenosis, a fetid muco-purulent discharge, and a dull pain extending to the cheek, eye, and forehead. "On inspection, small, hard, but elastic tumors, multiple and arranged in clusters, are noticed, more frequently on the septum than on the turbinated bodies or the floor of the nose, together with large masses of greenish or brownish dry scabs of hardened secretions, which when removed forcibly disclose a bleeding ulcerated surface with raised, indurated edges."[1]

A comparison with the descriptions of tuberculosis and syphilis gives the diagnosis between them.

The TREATMENT and PROGNOSIS are the same as for lupus occurring in other parts.

Carcinoma of the Nose.

Malignant disease rarely attacks the nose. When it does it is usually primary and sarcomatous, epithelioma and carcinoma being very uncommon. The septum is the most frequent place of origin. The tumors are usually single and unilateral. A malignant tumor first appears as a small pimple. Growing rapidly, it shows a broad base; it is varied in color and soft, bleeds readily, and when advanced tends to ulceration. There is a fetid greenish discharge. The symptoms are similar to those of fibroma (p. 37). The peculiar lancinating pain of cancer is felt. The adjacent glands are early involved. The appearance and history of the tumor, and the sensation on probing, generally make the diagnosis easy.

The PROGNOSIS and TREATMENT are, as in cancer elsewhere, most unfavorable. Early and radical surgical extirpation affords the best chance of arrest. In small, limited tumors the cold snare with cauterization of the base is the best treatment. In the case of more extensive growths the operations come within the domain of general surgery.

THE NASO-PHARYNX.

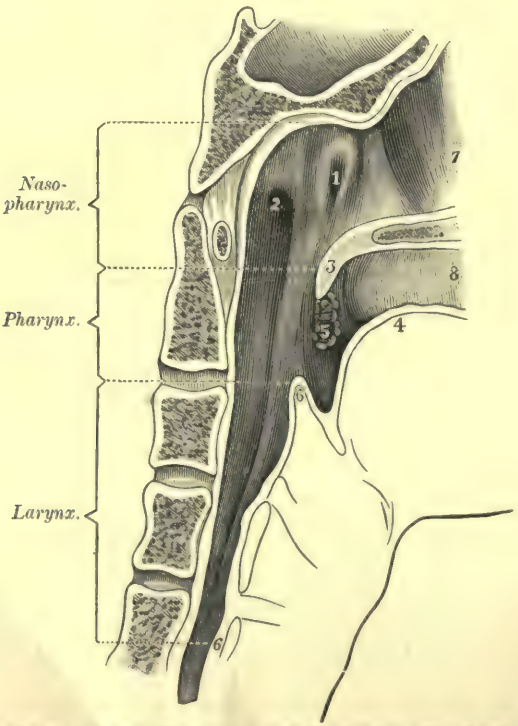
STRUCTURE.—The structure of the naso-pharynx, which is the upper part of the pharynx, extends from the basilar process of the occipital bone down to the lower edge of the soft palate. The pharynx continues downward to the greater cornu of the hyoid bone under the name of the oro-pharynx, and then extends to its termination at the lower border

[1] Carl Seiler, M. D., Burnett's *System*, vol. ii. p. 25.

of the cricoid cartilage, this part being known as the laryngo-pharynx. The further continuation of this tube in its course to the stomach is called the œsophagus. The pharynx is a membranous tube flattened from before backward and slightly concave in front. It is between four and five inches in length, and consists of a very tough fibrous framework to which is attached a complex muscular system. It is lined with mucous membrane and supplied with nerves and bloodvessels.

The naso-pharynx is the widest portion of the pharynx; it communicates in front with the nose, at each side with the ear by means of the Eustachian tubes, and below with the mouth through the oro-pharynx. It resembles a carriage hood in shape, is quadrilateral in form, and widest from side to side. Its roof or vault is formed by a portion of the posterior part of the body of the sphenoid and the basilar process of the occipital bone. It is bounded in front by the oval open-

FIG. 3.



Sectional view of the pharynx (Browne): 1, left Eustachian tube; 2, left fossa of Rosenmüller; 3, palate and uvula; 4, tongue; 5, left tonsil; 6, 6, upper and lower boundary of larynx (epiglottis and cricoid cartilage); 7, cavity of nares; 8, cavity of mouth.

ing of the nasal fossæ; these are separated by the vomer, which on this surface resembles a ploughshare in shape, as its name indicates, being broad above where it articulates with the sphenoid, and sharp below at its junction with the hard palate. Behind are the vertebral bodies, reaching as far as the arch of the atlas, the wall curving forward as it ascends. The cartilaginous portion of the Eustachian tubes limits it laterally.

The pharyngeal orifice of the Eustachian tube is trumpet-shaped, and is about two-fifths of an inch across vertically and one-fifth transversely. Its edge is formed by a cartilaginous ridge, quite prominent behind and above, less so in front, and absent below. It is level with the floor of the posterior nares, and about one fifth of an inch below the base of the skull. It is open when the muscles of the fauces are in action and closed when they are at rest. Three muscles, the tensor palati, levator palati, and palato-pharyngeus, dilate the Eustachian orifices.

The *mucous membrane of the naso-pharynx*, which is continuous with that of the nose and oro-pharynx, also extends into the Eustachian tubes. It is composed of both fibrous and connective tissue with numerous glands. The epithelium is cylindrical and ciliated, and is mottled in color. The mucous membrane forms a fold which extends from the upper border of the Eustachian tube to the edge of the posterior nares, while from the tube's posterior border another fold extends to the back of the soft palate. There is a depression of variable depth called the *fossa of Rosenmüller* situated between the Eustachian apertures and the back wall of the pharynx. (Fig. 3 (2), p. 44.)

The *glands* are both conglomerate and follicular. The conglomerate are most abundant on the back of the velum and the posterior border of the Eustachian tubes; the follicular cluster on the vault forming the pharyngeal, third, or Luschka's tonsil.

Luschka's tonsil, which becomes atrophied in adults, is about one fourth of an inch thick, sometimes extending over the entire width of the vault and on to the ridges surrounding the Eustachian tubes. It has the appearance of a soft cushion, fissured, and with rounded eminences. There is frequently a small opening in the middle, leading to a sac or bursa.

The *blood supply to the naso-pharynx* is from branches of the internal carotid. The veins form a deep plexus in the deeper layers and empty on to the internal jugular. The lymphatics, which are arranged in a network in the muscular and mucous layers, end in the glands at the base of the skull.

The *nerves* are derived from the second division of the fifth, with a few branches from the glosso-pharyngeal and vagus.

FUNCTIONS.—The functions of the naso-pharynx are for the most part the same as those of the nose. For this reason any change in the shape or any interference with its space modifies breathing, smelling, hearing, speaking, or singing. It is believed that the secretion is of service in lubricating the pharynx when food is swallowed.

ACUTE NASO-PHARYNGITIS.

DEFINITION.—Acute naso-pharyngitis is an acute inflammation of the mucous membrane of the vault of the pharynx. It is most frequent in adults. The exciting cause is exposure to cold. The predisposing cause of most of the attacks is probably, as in the nose, a previous chronic catarrh.

SYMPTOMS.—The onset of the attack is usually sudden and attended with much fever and malaise, a hoarse metallic voice, great dryness and

burning in the roof of the mouth, with pain on swallowing; which symptoms often persist for several days, accompanied by nausea and constipation. This condition is followed by a profuse thick grayish muco-purulent discharge which flows into the pharynx, increasing the digestive disturbance. Pain is usually present from the first, extending from the vault toward the angles of the jaws, with a soreness in the muscles of the neck and an itching about the fauces.

There is generally more or less obstruction of the Eustachian tubes, though autophonia is sometimes present.

The DIAGNOSIS is made from the foregoing described symptoms, by posterior rhinoscopy, and by the absence of inflammation in the nose or of white spots on the tonsils.

Acute naso-pharyngitis usually lasts somewhat longer than an acute rhinitis, and there is apt to be more prostration.

TREATMENT.—A hot mustard foot-bath, with a full dose of quinine and calomel at bedtime, and a saline purge in the morning, aconite for pain (tablet triturates of aconitia, gr. $\frac{1}{100}$, for about four doses), and phenacetin, gr. x, for headache, with hot saline douches, either nasal or post-nasal, followed by powdered acetanilid or boracic acid blown on the membrane, are usually the best remedies. Syrup of hydriodic acid, ʒj t. d. p. c., is often beneficial.

CHRONIC NASO-PHARYNGITIS.

DEFINITION.—This disease is usually known as naso-pharyngeal catarrh. It is characterized by a thick muco-purulent secretion from the glandular structures in the pharyngeal vault, which, dropping down into the pharynx, gives rise to much irritation.

ETIOLOGY.—The primary causes are necessarily obscure, but the views put forward by Bosworth appear most reasonable. They are briefly as follows: "In youth attacks of inflammation of the glandular structures in the vault result in hypertrophy (adenoid disease), which, naturally atrophying in adult life, assumes a desquamative process, which is the source of a muco-purulent discharge. Or a muco-purulent catarrh may arise without any preceding hypertrophy, probably a frequent cause of which is an attack of one of the exanthems, notably scarlet fever or measles." After its onset "the disease should be regarded in all cases as a purely local condition, and not dependent on any general dyscrasia." He considers, further, that the chronic inflammation gives susceptibility to cold-catching, and is therefore a cause and not a result. Tobacco may aggravate, but does not originate, the disease. Abuse of alcohol is undoubtedly a prolific cause, probably by reason of the intimate relations between the stomach and pharynx; thus, an alcoholic gastritis may give rise to a sympathetic pharyngitis. However, the chief causes are diseases of the nose, which interfere with nasal respiration, and, thus disturbing the function of the pharyngeal tonsil, produce irritation. The air entering from the diseased nose is dry instead of moist, and therefore the clear white fluid which normally flows from the pharyngeal tonsil is thick and tenacious, and irritates the pharynx, especially through the efforts made to dislodge it. Consequently, pharyngitis sicca is purely a symptomatic disease and a variety

of naso-pharyngeal catarrh. Tornwaldt's bursa Bosworth regards as developed from an hypertrophied tonsil in such manner that it forms a cavity in which the discharge is pent up.

DIAGNOSIS.—Posterior rhinoscopy reveals either an enlarged pharyngeal bursa or a broad diffuse hypertrophy of the tonsil. An enlarged pharyngeal bursa appears as a "rounded, almond-shaped projection in the median line and about midway between the prominence of the atlas and the dome of the pharynx." Instead of this we may see a general diffused hypertrophy of the pharyngeal tonsil, and either condition is characterized by a thick, stringy, muco-purulent secretion.

PROGNOSIS.—The disease is not dangerous, nor does it tend either to increase or to get well spontaneously. It is probably not, writes Bosworth, "an efficient cause of laryngeal or bronchial disease, asthma, or deafness." It can be cured, but treatment usually requires much time and patience.

TREATMENT.—Internal medication probably has no influence except as it may improve the tendency to heal. But the restraint of alcoholism and the regulation of the digestion are important aids. Smoking need not be interdicted unless the patient finds his symptoms aggravated by it. The vault must be thoroughly cleansed twice a day with a warm antiseptic alkaline wash, applied with the post-nasal douche. This treatment must not be used if there is an obstruction, for fear of forcing fluid into the ears. Twice a week, at the office, after cleansing, astringent and stimulating applications should be made to the vault with a curved cotton-covered applicator carried up behind the palate, nitrate of silver, 20 to 30 grains to the ounce of Lugol's solution, with an equal quantity of glycerin, being particularly valuable. It is often best to destroy the bursa with the galvano-cautery, which for use in this situation is to be preferred to other caustics. Sometimes the patient's pharynx is so sensitive that it does not tolerate treatment through the mouth, the palate also being often too irritable to be drawn forward with a retractor or tied up with tapes. Under these circumstances the applications should be made through the nares.

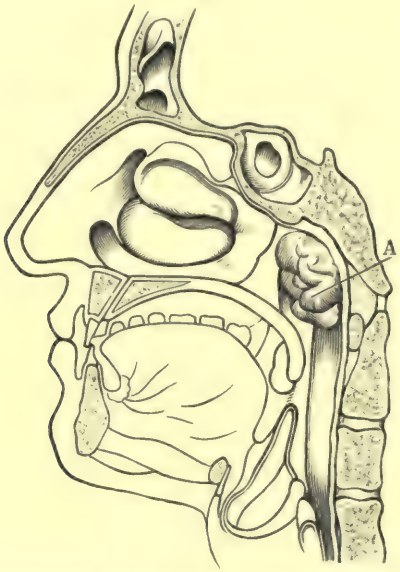
When the hypertrophy is much diffused curetting is preferable to the cautery. After burning, the parts should be douched. Cocaine must be applied before any operation.

ADENOMA.

DEFINITION.—Adenoma in the vault of the naso-pharynx is a true hypertrophy of the normal lymphoid structure of the pharyngeal tonsil. It is a disease of childhood, and may be congenital; it follows the rule of other glandular hypertrophies, tending to disappear at puberty, but up to the age of twenty-five years these growths are sometimes still large, and occasionally remain so, in a lessened degree, till the age of fifty is reached. After twenty the obstruction and inconvenience are not so great, but there exists an obstinate naso-pharyngeal catarrh. Nasal disease is present in about 50 per cent. of the cases, and in a lesser proportion there is some hypertrophy of the lingual tonsils. Fibrous structure is often found interlacing the adenoid mass, and this is more marked in the older patients. The growth may be large enough to fill

the naso-pharynx, or so small that its character is not readily apparent. It may grow not only from the vault, but from the walls, or it may spring entirely from the posterior wall. The growths are sometimes seen in scattered prominences or clustered into a well-defined tumor.

FIG. 4.

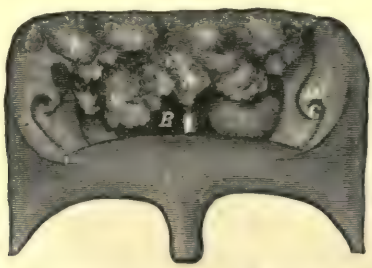


Position of adenoid enlargement as commonly located in the upper pharynx (Delavan).

ETIOLOGY.—The causes of adenoids are obscure: heredity appears to have an influence, and probably scrofula does also, and they are apt to appear after attacks of the eruptive fevers.

SYMPTOMS.—The chief of these is due to the obstruction of the

FIG. 5.



Adenoid hypertrophy at vault of pharynx (Lefferts).

posterior nares, which causes mouth-breathing. As a consequence, there is usually an open mouth, a vacant look, half-closed lids, pinched nostrils, prominent teeth, a high-arched palate, a pigeon breast, nasal catarrh, a dead voice as from a cold in the head, and, if singing is attempted, the upper register especially is muffled. Dulness of hearing,

and even of intellect, is often observable. Snoring is usual, and in infants there is often inability to nurse. Nervous reflexes, such as laryngismus stridulus, which probably always springs from this cause, headache, and cough are apt to develop, and the secretion from the naso-pharynx, being swallowed, may give rise to dyspepsia.

Kayser[1] points out that the reason that the difficulty in breathing through the nose is often out of proportion to the size of the adenoids is because most of the inspired air enters the naso-pharynx from the upper regions of the nose. The cough is sometimes so severe and paroxysmal as to simulate pertussis. That the cough is not due to tracheitis can be demonstrated by the fact that pressure on the trachea does not start it.[2]

Delavan[3] says: "One effect of the obstruction to nasal respiration is worthy of special consideration—namely, the permanent deformity of the bony framework of the nose and hard palate which generally accompanies it. While with the angular upper jaw and high-arched palate it is sometimes possible to find a normal nasal septum, the contrary condition is the rule." He goes on to speak of the nasal obstruction, when it exists during early life, as causing imperfect development and asymmetry of the osseous structures of the nose.

Diagnosis should be confirmed by anterior and posterior rhinoscopy: the latter is often difficult, especially in the case of children. The index finger, having been cleaned antiseptically and being protected by a shield, should be inserted behind the palate, when a tumor of a soft, velvety character will be detected; it is prudent to feel, at the same time, for the pulsation of anomalous arteries. The contraction of the velum upon the finger may be mistaken for a tumor, but these growths always lie behind, and, if the finger is pressed up, as suggested by Lennox Browne, and the lower portion of the septum is felt and traced up, the tip of the finger will impinge upon the growth, and the exploration can be completed by feeling the right and left lateral walls.

Anterior rhinoscopy generally shows an absence of serious nasal disease, but the passages are seldom sufficiently open to allow of the growths being seen. Posterior rhinoscopy, to be of service, requires that the palate should be drawn forward with a retractor and the tongue depressed. "Instead of the rounded, dome-like cavity of the pharynx, a round mass of a reddish gray tinge, with a mammillated surface, hanging down, as it were, and obstructing the view into the nasal cavities, is seen."[4] Bosworth further points out that "even a comparatively small growth will interrupt the view and shadow the broad upper portion of the nasal septum." In early life the surface resembles that of the brain; later it is smoother in contour.

Treatment.—Except in a few cases the only efficient treatment is the removal of the growths by surgery. Contraindications to operating are acute catarrhal conditions, hæmophilia, and anomalous arteries. In case of the presence of an epidemic in the house it would also be unadvisable to operate.

[1] *Archives of Otology*, N. Y., Jan., 1892.
[2] Lubet-Barbon, *Revue mensuelle des Maladies de l'Enfance*, Paris, Nov., 1895.
[3] D. Bryson Delavan; see *System of Surgery*, vol. i. p. 77, Lea Brothers, 1895.
[4] Bosworth's *Diseases of the Nose and Throat*, p. 553.

Fibroma.

Fibroma is a neoplasm formed of fibrous tissue, and is highly vascular. It grows from the bony roof of the vault, probably, as a rule, from the periosteum of the basilar process, and gradually extends downward into the pharynx and forward into the nose. It is a sessile growth, but is not a polyp, as it is sometimes miscalled, for it does not, in growing, preserve a narrow neck at its base; its attachments broaden in proportion to its growth, and the tumor may develop widespread adhesions to the neighboring parts. While not in itself malignant, it may do great injury to the adjoining cavities by filling them and crowding out their walls; thus, by pressing out the nasal bones, it gives rise to the characteristic appearance known as frog's face. It grows freely up to the age of twenty, after which it slowly atrophies.

Etiology.—Its causes are unknown. It will develop from apparently normal tissue in a healthy individual, usually of the male sex.

Symptoms.—These appear early, bleeding being among the first to show itself; this is apt to become severe and difficult to arrest. There is also much local irritation, with a persistent catarrh. Fibroma gives rise to the various symptoms of nasal stenosis as well as deafness and earache. Its extension into the pharynx causes attacks of suffocation and vomiting and interferes with eating. There is a copious mucopurulent secretion, which often becomes inspissated, and therefore very troublesome to dislodge.

Diagnosis.—Fibroma is unlike any other neoplasm. It is seen, in its early stage, as a single, smooth, rounded, irregularly shaped, dense, resisting tumor of a pinkish white color, and its surface is often streaked with large vessels. It is not so movable as an osteoma or a chondroma, and is much less so than an adenoma or a polyp. The needle or the probe reveals the difference in its density from all of these, while its ready and often serious tendency to bleed when examined with the forefinger is characteristic. It has no resemblance to a malignant growth, but sometimes the microscope alone can decide between fibroma and a fibro-mucous polyp.

Prognosis is favorable while the growth is still limited to the naso-pharynx, so as to admit of removal by the natural passages, but when it has spread farther operations are peculiarly difficult and the prognosis is grave.

Myxo-fibromata.

These tumors partake of the characteristics of both polypi and fibroma; they grow from the upper borders of the choanæ. They do not develop to a large size, and their symptoms are not formidable, being due to a certain amount of nasal stenosis, interference with deglutition and phonation, and slight catarrh. They may grow to a considerable size, but seldom extend beyond the naso-pharynx. They are not uncommon, rarely recur, and seldom bleed, and are best removed through the nostrils with the cold snare.

Chondroma; Sarcoma; Carcinoma.

Chondroma is an exceedingly rare disease. Bosworth found reports of only two cases.

Fortunately, sarcoma of the naso-pharynx is quite rare. The tumor may be either lobulated or pedunculated. The symptoms resemble those of fibroma, but there is also severe stabbing pain, often felt in the ear and worst at night, with dyspepsia and general cachexia. The appearance of sarcoma is frequently not sufficiently characteristic for a diagnosis, which must then be decided by a microscopical examination. Early removal with the snare, preferably the cold snare, affords practically the only chance of arrest.

Carcinoma is still rarer than sarcoma, to which its course and symptoms are very similar. Infiltration of the tissues of the neck occurs comparatively early.

The TREATMENT varies with the case, and the PROGNOSIS, except in very early operations, is almost hopeless.

DISEASES OF THE LARYNX.

Functions of the Larynx.

THE peculiar function of the larynx is that of phonation, although it also assists other portions of the tract in that of respiration. Phonation is effected by partial approximation of the vocal cords, so that the chink of the glottis is narrowed, and the expiratory current of air forced through this contracted opening causes the cords to vibrate; these vibrations are communicated to the escaping column of air, which the movements of the tongue, fauces, and lips convert into articulate speech. The vocal pitch is regulated by the degree of tension of the cords, being highest when they are most tense. The quality or timbre of the voice is due to the general contour of the upper respiratory tract. The vibrations set in motion by the cords are communicated to the air in the pharynx, mouth, and nose, and thereby the resonance of the voice is increased and its tone modified. To produce an agreeable voice the waves of sound must pass equally through the mouth and nose. When the palate is drawn too far up against the back of the pharynx the sound issues through the mouth alone, or when it hangs too low the sound-waves escape mainly through the nose. As has been stated in discussing adenoma of the vault, the bony dome of the naso-pharynx, when unobstructed, adds much to the resonance of the voice.

For these reasons it will often be found that vocal deficiencies, particularly in singing, are due to disorders in the upper air passages, and not, primarily at least, to laryngeal disease. It is therefore important to examine these parts, as well as the larynx, in all vocal difficulties.

Methods of Examination.

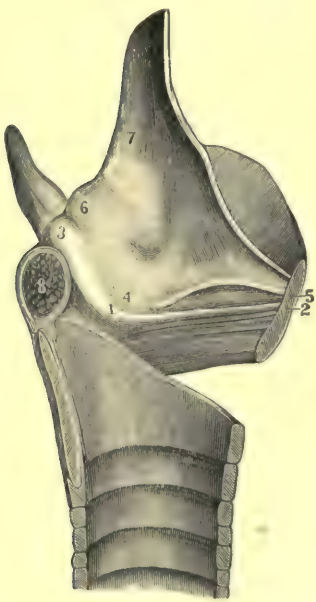
The laryngoscope, the light, the chair, etc. are described in the article on the Nose (p. 20), and therefore need not be repeated.

When the patient is seated and the light properly adjusted, he is requested to stretch his neck upward and throw his head back, and then to open his mouth and protrude the tongue as far as possible, making the effort to touch his chin with the tip; at the same time, with a napkin

in hand, he gently but firmly grasps the end between his thumb and forefinger. To procure the best view it is sometimes necessary for the examiner to hold the tongue himself, but most patients are readily taught to hold out their tongues efficiently, and this leaves the physician the freedom of both hands for other use.

The physician now seats himself opposite his patient with his reflecting mirror properly adjusted on his head or on an arm of his lamp, as described for rhinoscopy, so as to throw the light upon the soft palate where the velum joins with it. A large-sized throat mirror, previously warmed, is then taken, preferably in the left hand, held like a pen, and introduced into the mouth with the back toward the hard palate, till it touches the uvula, when it must be turned horizontally and gently pushed upward and backward so as to lift the uvula out of the way and yet not touch the back of the pharynx, which would cause gagging. The handle must be held in the right corner of the mouth to allow of a good view. In introducing the mirror care must be taken not to touch the tongue or to strike the teeth. It is better to repeat the inspection several times than to cause nausea by keeping the mirror long in the mouth.

FIG. 6.



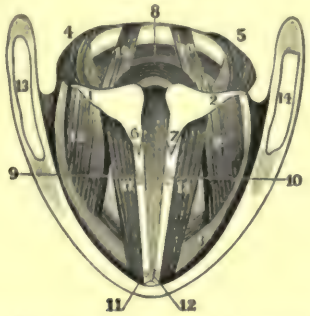
Side view of the larynx, showing the left ventricle of Morgagni and the left ary-epiglottic ligament (Browne): 1, 2, left vocal cord; 3, elevation indicating the site of the left cartilage of Santorini; 4, 5, 2, 1, entrance to left ventricle of Morgagni; 4, 5, left ventricular band (false vocal cord); 6, elevation indicating the site of the left cartilage of Wrisberg, with the cuneiform cartilage running down to 4; 7, aryteno-epiglottidean (ary-epiglottic) ligament; 8, arytenoideus muscle.

When the mirror is in position the patient should be requested to take a deep breath and to make the sounds *ah*, *ur*, *ch*, or *ee*. A clear view of the larynx is usually obtained: it may, however, in certain cases require several attempts at the same or during future sittings before this is accomplished satisfactorily. The movements of the examiner must be quick, decisive, and, above all, gentle; and the patient must make a mental effort to resist gagging, and must not pull away, but hang his tongue out as far as possible, and, grasping it firmly and gently, steady it in that position. Sometimes it is necessary to overcome the sensitiveness of the pharynx and palate by mopping them first with a 4 per cent. solution of cocaine just where the mirror should rest. When the patient is a child laryngoscopy is often difficult without an anæsthetic, but only occasionally impossible. When the mouth is not voluntarily opened compression of the nares will accomplish the desired end. If the tongue is not protruded, depressing it firmly with a spatula will often afford a view of the larynx, which is higher up in a child than in an adult. It is possible that it may be necessary to cause gagging, and then a rapid view of the larynx may be obtained.

The Laryngoscopic Image.—In the upper portion of the field will be

seen the yellow leaf-like epiglottis attached to the base of the tongue and extending on either side to the arytenoids, which appear as small rounded knobs; in the lower portion of the field are the two folds of mucous membrane called the ary-epiglottic folds, forming the lateral borders of the laryngeal tube; below these are seen the ventricular

FIG. 7.



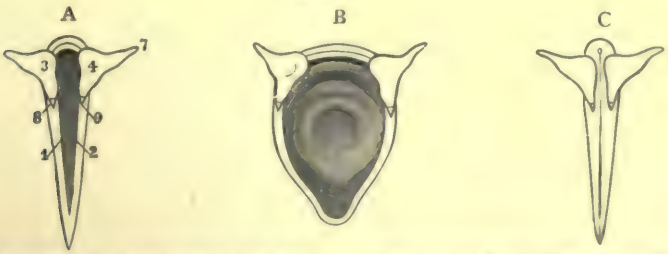
View of a section of the larynx from above (Browne): 1, 2, processi musculares of the arytenoids; 3, 3, cricoid cartilage; 4, 1, and 5, 2, posterior crico-arytenoidei muscles; 6, 7, processi vocales of arytenoids; 6, 11, and 7, 12, vocal cords; 8, arytenoideus muscle; 9 and 10, Elsberg's vocal nodules; 11 and 12, sesamoid cartilages; 13 and 14, thyroid cartilages; 15, 16, crico-arytenoidei lateralis muscles; 17, 18, thyro-arytenoidei muscles; 19, 20, crico-arytenoid ligaments.

bands, which are folds of mucous membrane with ligament (thyro-arytenoid) imbedded in their free edges.

Immediately beneath these appear the openings of the ventricles of Morgagni as thin black lines, but by turning the mirror laterally their pouch-like form appears.

On a still lower plane are seen the vocal cords, showing up brilliantly

FIG. 8.



A, glottis in repose: 1, 2, vocal cords; 3, 4, section of the arytenoid cartilages; 5, elastic band; 6, 7, processi musculares of arytenoids; 8, 9, processi vocales of arytenoids; *B*, glottis in deep inspiration; *C*, glottis in the production of tone.

white and glistening against the pink walls of the larynx. On phonating a mere chink is seen between them, but on deep inspiration the space spreads out to a triangle. The muscles rotating the angles of the arytenoids draw the cords together or apart. Between and below the cords are seen a part of the cricoid cartilage, the rings of the trachea, and, exceptionally, the bifurcations.

The normal color of the larynx varies from a deep red to a light pink; where the underlying structure is glandular, as the under surface

of the epiglottis, the ary-epiglottic folds, the ventricular bands, and the commissure, it is always a deeper red.

The edge of the epiglottis and the cartilages of Wrisberg and Santorini show up through the mucous membrane with a yellow hue. Any departure or irregularity of color and symmetry of outline, swelling, or deviation of the parts, or interference in the action of the cords, must be noted.

Acute Laryngitis.

Definition.—In accordance with the definitions laid down by Bosworth, diseases of which the name ends with "itis" are catarrhal affections, and do not cover phlegmonous or œdematous conditions. Acute laryngitis is therefore an inflammation which is mild, but acute. It is not serious in character, but it is annoying because it is usually accompanied by loss of voice.

Etiology.—The theory propounded by Bosworth with regard to like affections in the nose and naso-pharynx appears to apply with equal justice to the larynx—viz. that these acute attacks usually occur because there has been previously some slow-developing chronic condition as a predisposing cause which gives the exciting cause (a cold, etc.) a ground upon which to establish itself. Most laryngeal affections are due to some chronic disease in the upper air tract interfering with the proper heating or moistening of the air in its passage through the nose, or because of some nasal stenosis, or some destruction in the vault, such as adenoma, causing mouth-breathing, and so necessitating the ingress of raw air directly to the larynx; among the exciting causes are cold, dust, or irritating vapors, and over-use of the voice, gout or rheumatism, an attack of one of the exanthemata, and, in singers, the disorder is said to be often connected with enlargement of the glands at the base of the tongue.

Symptoms.—Dysphonia and cough, with more or less redness and œdema of the cords and the secretion of a tenacious, irritating mucus.

Diagnosis.—A laryngoscopic examination is often prudent, as the symptoms are similar to those of some graver affections. On inspection the whole mucous membrane is found to be bright red and symmetrically swollen, the cords being pink; the movements are not usually interfered with. The voice is hoarse and metallic, and the dysphonia begins suddenly. The attack usually runs its course in about a week.

Treatment.—If, as is usual, the upper tract is involved, treatment of that portion will usually be more useful than a direct treatment of the laryngeal inflammation. For such treatment see article upon the nose (page 25). A spray of Dobell's solution, followed by an astringent spray, such as nitrate of silver, gr. iij ad ℥j, is useful. In the early stage inhaling a vapor from compound tincture of benzoin floated on boiling water is remarkably soothing. An ice bag applied to the throat is also comforting, and the use of a demulcent lozenge, such as the paté de guimauve or the paté Aubergier, excites secretion and relieves the dryness. In the acute stage direct topical applications are too irritating. Codeine, gr. ¼ p. r. n., may be needed for the cough, and a saline purge in the morning is generally advisable. For the first forty-eight hours

confinement to a room in which the temperature is uniform and a little steam is allowed to evaporate is also generally wise.

In children this affection, when it does not extend below the glottis, calls for similar treatment, and the prognosis is equally favorable.

Acute Subglottic Laryngitis.

Acute subglottic laryngitis is attended with much more alarming symptoms, owing to swelling and consequent tendency to spasm and dyspnœa.

Etiology.—The immediate cause is generally a cold, but the predisposing cause is possibly a peculiar tendency to chronic inflammation of the lymphatic glands, the condition which leads to or stops short of scrofula, and it is the swelling of the lymphatic tissue, which is abundant beneath the glottis, which causes the dyspnœa.

Symptoms.—The attacks frequently return each winter or spring, and last about ten days; they are usually worse at night. The cough is croupy.

Diagnosis.—The larynx should be viewed, if possible, when, besides a general redness and swelling, there is a puffed-out look to the mucous membrane below the cords. In diphtheria the false membrane would be found in the fauces, and general septic symptoms would be present. In membranous croup the constitutional disturbance is greater and aphonia comes on earlier, the cough is weak and low, the advance is more even, and there are not such marked nocturnal exacerbations. The cervical glands may be involved in any of these diseases, but are generally most swollen in diphtheria.

The treatment is similar to that in supra-glottic laryngitis. The child must be kept in one room where a little steam constantly evaporates. Dover's powder is sometimes needed at night; calomel, gr. i–ij, in broken doses, is valuable. Ammonium chloride, gr. v, given t. d. with mistura glycyrrhizæ co., ℥j (adult dose), loosens the secretion.

During an attack a bath at 100° F. is often effective; inhalations of steam with a few drops of chloroform, or hot compresses with a little mustard put round the neck, serve to mitigate the symptoms; causing the patient to gag by passing the finger into the pharynx will often dislodge the phlegm and avoid the depressing effects of emetics.

Chronic Subglottic Laryngitis.

This is a rare and dangerous disease in which there is hyperplasia in the tissues below the glottis, causing stenosis and dyspnœa. "It is probably," says Bosworth, "in most instances a form of lymphatic hypertrophy." It most often appears between the ages of fifteen and twenty-five, and more frequently in females than in males.

Etiology.—It is generally associated with scrofulosis, or at least with a tendency to lymphatic enlargements. These diatheses would appear to be the chief predisposing causes. Rhino-scleroma, with which it is sometimes associated and which it resembles, may be another cause, as also may severe attacks of simple chronic subglottic catarrh. It has been observed as a sequela to attacks of typhus and typhoid, so that these may be looked upon as occasional exciting causes.

Symptoms.—Its course is insidious and slow, attended with an advancing aphonia, which may become complete, and is usually due to the hypertrophies interfering with adduction of the cords. Cough is not apt to be present until late in the course, when it is generally severe, particularly if there is an intercurrent catarrh giving rise to dyspnœa and the outpouring of a viscid tenacious secretion. In this event, more particularly, the cough may be croupy or metallic in character and have a resemblance to that of pertussis.

Diagnosis.—These symptoms readily point to its seat, which a laryngoscopic examination should confirm. It will then be seen that the appearance is that of an ordinary chronic laryngitis above the glottis, while below the vocal cords will appear two rounded or oval-shaped masses which project from the sides of the subglottic portion of the larynx beyond the inner margin of the vocal cords. These masses are bilateral, symmetrical, opaque, and gray or faintly reddish in color, and they are firm and unresisting to the touch. Usually the cords can readily be abducted, but are adducted with difficulty. The disease can easily be distinguished from perichondritis, which affection it most nearly resembles because it is not irregular and unilateral.

The prognosis is unfavorable.

Treatment.—Simple local applications are not of use. Dilating with O'Dwyer's tubes has proved fairly successful. Tracheotomy has sometimes to be performed, after which the cauterization of the thickened tissue has been carried out through the wound. Internal administration of iodide of iron, 10 to 20 gr.; t. d. p. c., is strongly endorsed by authorities. It is possible that injections with a syringe directly into the trachea might be of service.

Chronic Catarrhal Laryngitis.

This affection is a chronic inflammation of the laryngeal mucous membrane, purely catarrhal in character. While it causes more or less dysphonia, it may exist without much discomfort and with little interference with the ordinary use of the voice. It is a common disorder, particularly with those who make much use of their voices; it is not a dangerous malady, is more frequently found in males than in females, and appears in early manhood and middle life rather than in youth or in age.

Etiology.—Chronic laryngitis is almost invariably accompanied by some disorder of the upper air tract, which is probably, with few exceptions, its primary cause, for similar reasons to those given as predisposing to acute laryngitis. It is also, as stated, most common among those who make excessive use of their voices, and therefore overwork of the organ is a frequent exciting cause. Alcoholics often suffer from it, probably not often primarily, but because the laryngeal affection is a part of the chronic inflammation that so frequently exists throughout the length of the respiratory tract in those who have for a long time been intemperate in the use of alcohol. It is also often associated with a gouty or rheumatic diathesis. Excessive smoking, while it is usually irritating to an existing laryngitis, especially if it be in an acute stage, is probably not a primary cause. The inhaling of dust or irritating vapors may be an exciting cause.

SYMPTOMS.—There is always some vocal disability, varying greatly in amount, and generally a husky voice. A tendency to frequent hawking of mucus and a slight occasional hacking cough are also symptoms. The throat is usually at its worst in the morning, clearing up at midday, and getting tired and husky again at night. There is often a feeling of dryness, fatigue after using the voice, and occasionally a slight pain. The effort of speaking in public or of singing sometimes results in complete temporary aphonia, but it is not unusual for the voice to be clearer and stronger during the excitement attending the exertion, and to fail more markedly when repose comes.

DIAGNOSIS.—The laryngoscope reveals a reddened mucous membrane with the bloodvessels injected, and usually a moderate, uniform, and symmetrical swelling, especially noticeable in the commissure and the ventricular bands. The cords are generally of a grayish and not infrequently of a pinkish color, and their adduction is often imperfect. There are occasionally slight erosions, and sometimes proliferations are found in the commissure. The secretion is not apt to be abundant, but is dry and tenacious. The symptoms and appearances may be very similar when a foreign body has been lodged for some time in the larynx or when a growth exists. A tumor would be unilateral, which is a rare condition in catarrhal swellings. Tubercular laryngitis, unless chronic laryngitis also exists, which is not infrequently the case, may usually be distinguished by the anæmic color of the membrane and the irregularity in form and disposition of the swellings. If these appearances exist, an examination of the lungs should be made, though it must be remembered that tubercular infiltration of the larynx often occurs with pulmonary signs too slight for any but the most skilled auscultators to detect. The writer believes that primary tubercular laryngitis is exceedingly rare, and that when the lungs give no sign the disease commonly exists in the bronchial glands. Syphilis of the larynx sometimes resembles simple chronic inflammation, and can only be diagnosed by evidence of the disease in other parts, from the history of the patient, or by the test treatment with iodides.

PROGNOSIS.—There is but little tendency toward recovery except through treatment, which must almost invariably be directed toward curing the accompanying nasal or pharyngeal disease. With proper treatment the prognosis is good, though a complete cure is not common. Chronic laryngitis is doubtless a frequent predisposing cause of neoplasm, but probably not of cancer or tuberculosis.

TREATMENT should be first and especially directed toward cure of disease in the nose and naso-pharynx, and the writer has seen several cases in which the laryngeal inflammation has entirely disappeared, with only placebos applied to the larynx, as soon as the disease in the upper respiratory tract was removed. In cases where the larynx is irritable and where there is but little hyperplasia, and no erosions or vegetations, it is best to spray first with Dobell's or a similar solution, and to follow this with a mild astringent spray, such as a 4 per cent. solution of ichthyol.

Where there are erosions or chronic thickening it is often advisable first to spray in a 4 per cent. solution of cocaine with an equal part of a 10 per cent. solution of antipyrine, and in about four minutes to

lightly apply on a cotton swab a mixture of one part of Lugol's solution of iodine with three parts of glycerin to the erosions or swellings. The patient should draw out his tongue while the physician, holding the throat-mirror in place with his left hand, rapidly makes the application. This may need to be repeated four or five times at the rate of two treatments a week. For vegetations spraying with pure alcohol or its direct application on cotton will often cause them to disappear. Should these or the erosions remain obstinate, a slight curetting with a single curette, followed by an application of pure lactic acid, is effectual and not painful if a 20 per cent. solution of cocaine is first painted on with a cotton applicator. In some obstinate cases the use of the globe inhaler charged with the benzoin vapor is very valuable. This may have to be continued daily for from one to three weeks. The systemic treatment must be governed by the general condition of health; dyspeptic and hepatic symptoms often need attention.

Trachoma Laryngitis.

Definition.—This disease was first known as chorditis tuberosa. It is characterized by the presence of small nodules upon the vocal cords midway between their anterior and posterior extremities. These nodes are sessile, projecting out from the free edges of the cords. After fully developing they cease to enlarge. They sometimes appear on both cords at once, always just opposite each other, or the second may develop later, but still always in the same relative position as the first nodule.

These nodules seem to be simply hypertrophies of the fibrous tissue of the cord. It is probable that they only occur as a consequence of overstrain in singing when there is an existing chronic laryngitis. As they are always limited to the posterior half of the larynx, it is possible that they are only developed by some special act of vocalization.

The only symptom is hoarseness, and this is especially noticeable when high notes are attempted. The execution of high notes often becomes impossible.

Diagnosis.—The laryngeal image shows a small grayish white nodule on one or both cords, always in the same position.

Treatment consists in rectifying the general laryngitis and in making stimulating applications directly to the nodes, repeated every other day until they disappear. They should first be touched with a 20 per cent. solution of cocaine, and then such a remedy as Lugol's solution or lactic acid should be applied with an applicator having a small end around which is twisted a very thin wisp of cotton, which must not drip or else the liquid will be pressed out and burn beyond the node.

Laryngitis Sicca.

Definition.—This is a catarrhal inflammation in which there is very little secretion and crusts adhere to the laryngeal mucous membrane; these crusts usually lie below the cords. It is often associated with, and is undoubtedly analogous to, atrophic rhinitis.

Symptoms.—The crusts are apt to accumulate in the night, giving rise to much irritation, with coughing and hawking and more or less

aphonia, upon awakening, which continues in a slighter degree throughout the day. Dyspnœa may be present and the expired breath is offensive. The secretion is of a greenish yellow color and is occasionally tinged with blood. The laryngoscope shows the existence of the crusts. The course is chronic, and it is apt, like atrophic rhinitis, to be intractable.

TREATMENT.—If disease exists in the upper portions of the tract, it should be treated, the laryngeal membrane washed by spraying with Dobell's solution, and then any remaining crusts carefully removed with a cotton swab. A solution of nitrate of silver or sulpho-carbolate of zinc, gr. x–xx, is applied twice a week. The patient should use a spray of one of Seiler's antiseptic tablets twice a day in the interval.

ACUTE PHLEGMONOUS LARYNGITIS.

DEFINITION.—By this term is meant a simple active inflammation of the laryngeal mucous tissues, and particularly the submucous tissues, accompanied by marked swelling and œdema. As it is essentially an acute cellulitis, it tends to the formation of pus and sloughs. This affection is often also called œdematous laryngitis, but, as Bosworth very properly says, the latter title should only be applied to a passive non-inflammatory œdema. It is not a common disease, attacks males more often than females, and is most apt to occur between the ages of twenty and thirty years.

ETIOLOGY.—The attacks most frequently come on after exposure to cold, but sometimes they follow overstrain of the voice, and are occasionally secondary to other inflammations of the tract, such as quinsy and glossitis. Iodism is also believed to be a cause. It sometimes accompanies erysipelas or it may develop as a localized erysipelas.

SYMPTOMS.—The attack usually comes on suddenly with chilliness and a temperature of 100° to 101° F. and within twenty-four hours dyspnœa develops. The breathing is rapid and stridulous both on inspiration and expiration, the color cyanotic, the countenance anxious, and the patient restless, and in three or four days death may ensue unless relief is obtained. Sometimes the disease is milder, and resolution takes place in thirty-six hours from the discharge of an abscess; in still lighter cases only a small unilateral abscess may form, in which event there is but little dyspnœa with some aphonia and dysphagia, and more or less pain and sense of fulness around the larynx. In children a cough is not unusual.

DIAGNOSIS.—This is most surely made with the laryngoscope. The mucous membrane appears inflamed and swollen, tense, semi-opaque, and of a bright red color. Palpation with the index finger enables the "rounded, partially resistant masses distinctive of phlegmonous infiltration of the larynx" to be felt. The suddenness of the attack and the early development of swelling, and consequent dyspnœa, serve to distinguish it from a disorder caused by a foreign body or from passive œdema.

PROGNOSIS.—The danger is from suffocation, and tracheotomy is often needed in severe cases, but usually within the first thirty-six hours, after which time, if this procedure is not called for, the swelling begins

to subside. In the more chronic and milder cases a discharge of pus generally brings relief.

TREATMENT should be begun promptly. Inhalations of steam with tincture of opium, compound tincture of benzoin or carbolic acid ʒj to hot water Oij, may be used, or more comfort is sometimes obtained from cold, ice being held in the mouth and also applied externally. A 4 per cent. solution of cocaine applied frequently to the larynx, and a calomel purge followed by a saline, may suffice for the milder cases, provided the patient rests in bed and ceases to use his voice. However, when the dyspnœa is considerable, the face of the epiglottis and the ventricular bands must be scarified with a concealed laryngeal knife or an ordinary curved bistoury, the blade being wrapped with cotton to within a quarter of an inch of the point; the knife must be guided by the finger preferably, or by using the laryngoscope. Scarification may have to be repeated three or four times in the twenty-four hours.

If these means fail and it is not possible to open the abscess, it becomes necessary to resort to tracheotomy or intubation. If the former be performed without a tube at hand, threads can be passed through each side of the wound and tied behind the neck, so as to keep the edges apart, as suggested by Delavan. As the danger point is often reached with great suddenness, the surgeon should be always within call and his instruments should be always ready.

ŒDEMA OF THE LARYNX.

Under this head are included only cases of simple œdema without inflammation or any other local lesion. In these cases there is a purely serous exudation without other changes.

ETIOLOGY.—In some persons there is a peculiar general paresis, frequently hereditary, which, by causing a local vaso-motor paresis, gives rise to œdema. Pressure upon the cervical veins by a tumor or aneurysm may cause it, and it sometimes results from iodism. It is stated to be occasionally present in the newborn as a consequence of degeneration of the placenta. The most usual cause is, however, an extension of a general dropsy.

SYMPTOMS.—These are similar to, but much milder than, those of acute phlegmonous laryngitis. The dyspnœa comes on more suddenly and is most noticeable on inspiration; there are no inflammatory symptoms.

DIAGNOSIS.—The laryngoscope reveals a grayish semi-translucent, swollen laryngeal membrane with an absence of inflammation.

PROGNOSIS.—There is danger of suffocation in severe cases unless the treatment is active and prompt.

TREATMENT.—When the œdema is due to renal or hepatic disease, the patient should be freely purged and diaphoresis induced. For the former purpose croton oil, minim 1, on sugar or in a capsule, is the most rapidly acting and efficient drug, and to produce the latter effect pilocarpine gr. $\frac{1}{8}$, injected under the skin, is best. In cardiac dropsy fluid extract of digitalis or the tincture of strophanthus, minim 1, subcutaneously, is often demanded. Any depression may be met with alcohol. The room should be warm and the air kept moist by the continuous

evaporation of steam. Measures similar to those advocated for the phlegmonous conditions are sometimes necessary to avert suffocation.

Croupous Laryngitis.

This term is defined by Bosworth as covering those cases in which there are none of the septic symptoms of diphtheria, but with which there exists a fibrinous exudation in the larynx. Formerly diphtheria of the larynx was miscalled croup; now croupous laryngitis is frequently miscalled diphtheria. A further discussion of this subject will be found in the article on Diphtheria. (See Pseudo-diphtheria, Vol. I. p. 707 et seq.)

Causes.—Croup of the larynx is probably dependent for its manifestations upon a specific germ, though this is not proved. It is of the same inflammatory character as croupous inflammation in other parts, resulting in the exudation of a false membrane; it is usually preceded by some inflammation of the lymphatic tissues of the fauces. In most cases the history is one of a sudden chilliness of the surface of the body. The attacks are limited to children before the age of puberty. The scrofulous, the mouth-breathers, and those subject to acute catarrhs, and boys rather than girls, are the most susceptible. Heredity plays some part as a factor. The disorder is feebly contagious, and appears to be neither endemic nor epidemic. It is a catarrhal inflammation with a profuse serous exudation: the fibrin of the exudation coagulates upon the surface of the inflamed mucous membrane, and retains, by means of its gummy consistency, the epithelial cells which are freely proliferated by this membrane.

Symptoms.—It is usually heralded by some signs of a cold, but often a hoarse, metallic voice is the first noticeable indication, though there is generally a chilliness followed by a rise in temperature to 102° or 104° F., with all the symptoms of a sthenic inflammation. The temperature remains high for from twenty-four to forty-eight hours; the throat is sore and dry; the larynx is tender to pressure; and there is pain on swallowing. More or less dyspnœa, which is most noticeable on inspiration, is present, and may increase with cyanosis, dilated nostrils, etc. until death ensues within a week from suffocation unless the membrane is happily dislodged. Albuminuria may exist, as it may also in other acute infectious diseases, especially in diphtheria.

Diagnosis.—The only disease for which croupous laryngitis is likely to be mistaken is diphtheria. In diphtheria there is always at some time a false membrane existing in the fauces. This membrane is yellow, efflorescent, and thick; it cannot be removed without causing bleeding, and becomes necrosed in twenty-four hours.

In croup also there is frequently, but not invariably, a false membrane to be seen in some part of the fauces, but it is of a bluish-white color, translucent and thin, and does not become necrosed. If, in addition to other methods of examination, a laryngeal image can be obtained (and this is seldom possible with children), the same characteristics are observed. While in diphtheria, as in croup, death may occur from suffocation, yet in the former the fatal result is more often due to septicæmia, which is never the case in croup. In the latter disease the

membrane may spread to the trachea and bronchi, in which event tracheotomy is usually resorted to, and resorted to in vain.

Treatment.—The exudation is often most beneficially reduced by full doses of calomel given frequently until the patient passes spinach-like stools. Alternately with the calomel, doses of tincture of the chloride of iron, ℥xv, with glycerin, should be exhibited.

Steam inhalations, particularly with lime water, are a valuable aid. Pilocarpine is sometimes of value, but is often depressing. Intubation is usually preferable to tracheotomy, and is required in the larger proportion of cases. (See Intubation, Vol. I. p. 686.)

Perichondritis.

It is probable that chondritis of the laryngeal cartilages never occurs except as a consequence of a preceding inflammation of the perichondrium. This inflammation may have been acute or chronic, and may lead to fibrosis or ossification. Fibrosis is rare, chronic in its course, occurs most often in strumous persons, and is probably not infrequently a result of chronic subglottic laryngitis. Acute perichondritis is a disease which is generally obscure and difficult to diagnose. Its onset is sudden; its symptoms are grave, and it usually causes considerable deformity of the parts.

Etiology.—Primary perichondritis is not uncommon. It may result from over-use of the voice or from simple catarrh. It is a disease usually secondary to syphilitic, tubercular, or malignant ulceration of the larynx. It may occur in the course of pyæmia, pneumonia, diphtheria, erysipelas, as a sequela of typhoid or one of the exanthemata. Disease of the cervical vertebræ, traumatisms, such as the impaction of a foreign body, are occasional causes. It is more common in males than in females.

Symptoms.—*The Thyroid.*—The inflammation is usually upon the inner face of one or other of the wings, in which event there is some difficulty in speaking, and there is dyspnœa caused by swelling. If the inflammation extends to both sides, this may be very serious, and the voice is then usually absent. An external swelling with tenderness and localized pain is diagnostic of involvement of the outer surface. When the entire thyroid is affected the other cartilages are almost invariably involved, and death is imminent from extensive necrosis. The disease is, however, generally unilateral. When suppuration occurs, giving rise to fistula in the median line, pus discharges either under the skin or into the laryngeal cavities.

Necrosis of the thyroid is usually less serious than that of the cricoid or arytenoid, because there is a readier outlet for the discharge of pus.

The Cricoid.—In this case the inner surface is always affected, causing symptoms of dyspnœa from swelling. The inflammation spreads rapidly to the mucous membrane of the lower larynx, interfering with the use of the voice through infiltration of the crico-arytenoidei laterales, and sometimes of the postici muscles. There is not much cough, nor do the symptoms change until hypertrophy or suppuration takes place. Bosworth says he has not known of a case of spontaneous resolution.

A sequestrum is generally formed, and fistulæ with discharge of pus may exist for years before it is finally expelled.

The Arytenoid.—The effusion usually takes place into the crico-arytenoid articulation, interfering with phonation by preventing movement of the cord. The swelling gives rise to dysphagia and aphonia. The necrosed cartilage is generally exfoliated much sooner than that of the thyroid or cricoid. The epiglottis does not necrose, but ulcerates, and this rarely occurs except as the result of preceding syphilis, cancer, or tuberculosis.

DIAGNOSIS.—The symptoms of perichondritis are those of acute inflammation, beginning suddenly with chilliness and fever (the temperature rarely rises above 101° F), loss of appetite, pains in the bones, and headache. These inflammatory symptoms with dyspnœa occur only in this disease and in croup and submucous laryngitis. Laryngoscopy shows acute inflammation spreading over the neighboring parts, with a swelling which is usually irregular and unilateral. The color of the larynx is translucent, grayish or light red. In submucous laryngitis the swelling is generally bilateral and symmetrical, while in croup its peculiar exudation is to be seen, if not in the fauces, at least in the larynx. The existence of inflammation distinguishes the swelling of perichondritis from that of a neoplasm, and the fact that the tiny whitish gray points scattered through the pink membrane are absent serves to distinguish it from tuberculosis. The part which is most swollen and inflamed indicates the seat of the cartilage which is specially affected.

PROGNOSIS.—These cases are usually chronic, and are apt to lead to laryngeal stenosis, and, except when secondary to some systemic disease, when all the cartilages are involved, they are not usually fatal. When the arytenoid is involved pus may sometimes burrow into the deep tissues of the neck, and the disease often extends to the cricoid. The necrosed cartilage is almost invariably expelled, and there generally remains voice impairment from an ankylosis of the crico-arytenoid joint.

TREATMENT.—The treatment must be prompt in the acute stage. Cold should be applied externally by means of Leiter's coil and pieces of ice kept in the mouth. Scarification of the endo-laryngeal tissues may be called for, or the application of leeches or wet cups. Abscesses must be freely and quickly opened, while care should be taken to allow the pus to be rapidly expelled from the mouth. An application of equal parts of Lugol's solution and glycerin to the laryngeal mucous membrane is sometimes of service. A cocaine spray is useful to relieve pain. When there is danger of dyspnœa intubation is generally preferable to tracheotomy. The prolonged use of iodide of potassium is to be recommended in most cases. This should be begun early and preceded by a mercurial purge. It is sometimes necessary to divide cohesions or to dilate strictures with bougies.

LARYNGEAL HEMORRHAGE.

Under this head we shall consider cases of hemorrhage exclusive of those dependent upon such causes as tuberculosis, syphilis, cancer, or the presence of a foreign body. Bleeding from the mucous membrane is not common.

Etiology.—The most frequent predisposing causes are undoubtedly certain general conditions, such as the spanæmia due to malnutrition, phthisis, anæmia, cardiac dilatation, hepatic cirrhosis, or the hemorrhagic diathesis. It has occurred in the onset of acute laryngitis, but for the most part it is rare in simple laryngeal inflammations; it may, however, arise from the superficial erosions of a chronic laryngitis, from violent coughing, vomiting, straining, etc., and has been occasionally noted in apparently healthy persons without any discoverable lesion.

Symptoms.—There is usually a tickling cough and a slight interference with phonation. Dyspnœa may be present on account of extensive submucous extravasation or the lodging of a clot in the glottis. The flow of blood is generally moderate, and it is raised in small particles unmixed with mucus or saliva. When there is only a slight oozing the blood may be retained for a time, and then appear in small dark clots.

Diagnosis is important in determining whether the blood has come from the lungs, larynx, or naso-pharynx. The laryngoscope must, therefore, be used and the upper air passages inspected. When the blood has come from the lungs, it is usually unmixed with mucus, and is seen coating the trachea, and there is generally some evidence of pulmonary disease. When the source is the naso-pharynx, blood can generally be seen in these cavities or trickling down the back of the pharynx. Sometimes the laryngeal hemorrhage takes the form of a hæmatoma, there being an extravasation beneath the mucous membrane without hæmoptysis. In this case the purplish rounded prominence is readily distinguished from œdema or neoplasm.

Prognosis.—The local condition is not a dangerous one, but may persist for weeks if it is resultant from certain general conditions.

Treatment.—The larynx should be sprayed at least four times in the twenty-four hours with an astringent solution, such as—

℞. Liquoris ferri persulphatis, ♏x;
Aquæ, ℥j.—M.

Or,

℞. Argenti nitratis, gr. v;
Aquæ, ℥j.—M.

Ice may be applied externally with benefit. The use of the voice and exercise must be interdicted. Hot fluids, alcohol, and tobacco should be forbidden. Codeine, gr. ¼, repeated as required, will generally control the cough most satisfactorally.

Syphilis of the Larynx.

A primary lesion is very rare, and treatment should be deferred until secondary manifestations appear.

Erythema.—During the second stage of syphilis, after the skin eruption has disappeared, erythema of the larynx may show itself. There is usually no pain or dysphagia, but the use of the voice is somewhat interfered with, particularly when the cords are involved. It is a round-celled infiltration of the mucous membrane, giving rise to swell-

ing and venous congestion, and it differs from a simple inflammation in the dark red, dusky hue and mottled appearance of the membrane, which is due to the fact that the turgid veins show beneath the epithelium.

TREATMENT.—The effects of specific treatment and the history of the case are needed to confirm the diagnosis. The disease usually yields quickly to constitutional treatment. The local irritation is best relieved by methods similar to those which are appropriate to a simple acute inflammation.

The mucous patch is seen very exceptionally on the upper surface of the cords, and still more infrequently on the epiglottis, the arytenoids, and the ventricular bands. The lesion consists of a round-celled infiltration of the epithelium. It is seldom followed by inflammatory changes, but may cause some loss of voice and slight pain on swallowing. It shows less tendency to occur, spread, or persist in the larynx than in the mouth or pharynx. Cauterization may be needed in addition to the specific general medication. The mucous patch appears as a small, grayish, slightly raised spot with a reddened areola.

A superficial ulcer is not frequent, but it may occur in the secondary stage several years after the initial chancre, and it usually develops from a superficial gummatous tumor or a mucous patch. Its progress is slow. There may be a slight impairment of the voice and a secretion of bloodstained muco-pus. Other symptoms are usually absent. It exhibits a characteristic tendency to recurrence. The ulcer is oval in shape and is but little depressed; it is yellow in color, with a scanty sanguineo-purulent discharge. The surrounding tissues are little if any inflamed. Being small, it is hard to distinguish when on the vocal cords, its most frequent seat, but in other parts of the larynx it is readily recognized.

TREATMENT.—A rubbing in of lactic acid, followed by applications of iodoform and glycerin paste upon alternate days, is the best method of local treatment.

The gummy tumor is a more common form of laryngeal syphilis than those already mentioned, but it seldom appears under from five to ten years after the primary lesion. Its seat is in the submucous tissues, and it may arise in any part of the larynx. "It varies in size from that of a pinhead to a large cherry."[1] It is a rounded, symmetrical tumor, which rapidly develops beneath a healthy mucous membrane. It may be single or multiple. In some cases after developing it remains without change, but much more commonly it soon breaks down into a deep ulcer. To avoid this disaster a test treatment with iodides should be used whenever a suspicious tumor appears.

The Deep Ulcer.—The deep ulcer is always the result of the ulceration of gummatous infiltration, which, as it is heralded by very slight symptoms, if, indeed, it be manifest at all, is not recognized until ulceration takes place. The ulcer is most frequently found first upon the epiglottis, second upon the vocal cords, then upon the ventricular bands, and last upon the commissure. There is more or less pain, tenderness, and impairment of function in proportion to the size and position of the ulcer. The ulcer rarely bleeds, but has a free muco-purulent discharge,

[1] *Twentieth Century Practice*, vol. vi. p. 387.

with which are mingled blackened shreds of necrosed tissue. As a later result, necrosis of one or other of the cartilages often occurs from extension of the infiltration of the perichondritis. Fresh gummatous infiltration is apt to take place, followed by ulcers; in healthy cases cicatrices are formed which may give rise to laryngeal stenosis. The appearance of the ulcer, with its punched-out edges, dark-red areola, and deeply-depressed surface coated with a copious flow of dirty yellow pus in which float blackened particles of dead tissue, is characteristic. The tuberculous ulcer, on the other hand, is not depressed; it is of a whitish gray color, with a scanty, ropy, mucous secretion. The edges are not so sharply defined, and the surrounding membranes are pale without an areola. Lupus is irregular in outline; the membrane is slightly injected and there is an absence of ulceration. The sarcomatous tumor may show some erosion, but no distinct pus-secreting ulcer. The ulceration of all carcinomatous tumors is distinguished by a ragged edge without an areola, and is apt to bleed. The cancerous cachexia generally exists, as well as considerable swelling of the cervical glands.

The PROGNOSIS is always uncertain. Although arrest is not infrequent, yet recurrence is very common, and, as already mentioned, cicatricial stenosis often results from the deep ulceration.

If the TREATMENT already spoken of for superficial ulceration is not succeeding, inhalations of corrosive sublimate, from 1 : 1000 to 1 : 500, have been found of service, but the important point is to push iodism to the verge of tolerance.

Cicatricial Stenosis.—Cicatricial stenosis is probably always preceded by ulceration, unless it is caused by œdema or a gummy tumor. The voice is always affected, the tone is harsh and characteristic, dyspnœa occurs both with inspiration and expiration, but is most marked with the former. Localized pain, tenderness, and difficulty in swallowing usually indicate extension of the deposits. Increased secretion and cough are not generally present unless there is also disorder below the larynx. The advance of the stenosis is usually slow and intermittent, being increased by frequently recurring catarrhal attacks. Its advance may demand immediate tracheotomy. The administration of iodide of potassium has but little effect, and is apt to cause iodism, and the condition, being one of stricture, has to be treated by dilatation, divulsion, or section. The use of Whistler's cutting dilator, followed by intubation, is usually the most effective method. The tube has sometimes to be worn continuously. The constitutional treatment of these affections is more particularly considered in the article on Syphilis, Vol. I. p. 895.

TUBERCULOSIS OF THE LARYNX.

Tuberculosis of the larynx appears most commonly in persons of a so-called tuberculous diathesis. It is especially found in acute tuberculosis. It has been proved that it occasionally occurs as a primary lesion, but the evidences, both post-mortem and clinical, show that there is in the large majority of cases a preceding pulmonary tuberculosis, and it is probable that in those cases in which auscultation fails to give signs of deposit in the lungs there is already an infection of the bronchial glands. The writer's belief is that in nearly all instances it is a

secondary infection from a thoracic deposit. The pathology of the affection demonstrates that the infiltration takes place, as a rule, in the submucous tissues, and that ulceration subsequently occurs as a result. The fact that tuberculous laryngitis very rarely arises as a secondary affection in Colorado, where a large number of cases of progressive pulmonary tuberculosis survive for unusually prolonged periods, tends to confirm this belief. Nevertheless, there are undoubtedly cases in which laryngeal catarrhal erosions have been infected by the tuberculous sputum, so that the disease in these instances has proceeded from the surface of the laryngeal membrane. The danger of tuberculous laryngitis lies not so much in the difficulty in curing the local disease, as in the fact that it is almost invariably allied with a tendency to the general dissemination of tubercle with an absence of self-limitation. With regard to the frequency of the occurrence of this complication the appended table will be found interesting:

Frequency of Laryngeal Tuberculosis among Cases of Phthisis—Larynx Involved.

No. of cases reported.				Per cent.
M. Mackenzie,	100,	larynx involved in		33.0
Louis,	193,	"	"	32.6
Heinze,	1226,	"	"	30.6
S. E. Solly,	250,	"	"	28.0

Laryngeal Ulceration.

M. Mackenzie,	100	13.0
Williglk autopsies,	1317	13.8
Schroetter,	723	6.0
S. E. Solly,	250	8.0[1]

These statistics would indicate that the percentage of the laryngeal complications in cases of phthisis is somewhat over 30 per cent., and of these ulceration occurs in less than 13 per cent.

SYMPTOMS.—The earliest symptom is usually weakness and lack of control of the voice, the tone often changing to a falsetto; the cause of this is generally infiltration in the commissure or around the arytenoid articulation, which prevents a full approximation of the cords, and it is probable there is often actual paralysis of the motor nerves. In some few cases there is a preceding or accompanying catarrh, in which event the voice may be harsh, as it also is when the infiltration extends to the posterior insertion of the cords. If the epiglottis or ventricular bands only are involved, there is little or no interference with phonation. Painful deglutition is generally an early and distressing symptom, and it usually, though not always, indicates the presence of ulceration. It is most marked when the epiglottis or upper portion of the arytenoids is involved, and there is often little or no pain attending affection of the cords or commissure. Pain is the chief cause of the fatal result in these cases, owing to the inability to take sufficient nourishment and its harassing effects. There almost always exists an irritating and frequent cough, especially during ulceration, when a thick, ropy mucus is con-

[1] "Report upon Cases of Tubercular Laryngitis treated in Colorado Springs," by S. Edwin Solly, *Therapeutic Gazette*, Nov. 15, 1893.

stantly secreted in the larynx in addition to the accumulation of the pulmonary sputa, which the partially paralyzed larynx has difficulty in discharging. There are pain and tenderness on pressure over the larynx. Necrosis of the cartilages from extending perichondritis is often present in the later stages, and it may give rise to sufficient œdema to cause dyspnœa, but not to the characteristic and extreme pain of perichondritis from other causes.

Diagnosis.—Weakness of the voice or hoarseness may, and frequently does, occur in the course of phthisis without being caused by tuberculous infiltration of the larynx, but if there is tenderness and dysphagia this complication almost certainly exists. The laryngoscope seldom fails to reveal the true condition to the practised eye. The special features of tuberculous laryngitis, particularly in the early stage, are usually rounded unilateral tumefactions and a bloodless and dry condition of the membrane, while the swellings over the deposits have a glazed surface of a pale grayish pink hue. The first deposit is usually in the commissure. When the arytenoids are involved it generally begins in one, but quickly spreads to the other, but when the ary-epiglottic folds or the ventricular bands are involved it is usually unilateral. The epiglottis is generally infiltrated along its margin, giving to it the characteristic appearance of a turban. There is very little hyperæmia to be seen in the larynx, but in a short time minute yellow points appear dotted over the area of the infiltrations. These points are tubercular nodules seen through the epithelium, and are usually the precursors of ulceration. The appearance of the tuberculous ulceration is worm-eaten, and has been compared to cut bacon. The ulcerations are shallow, and similar in color to the surrounding membrane, the infiltration going on so rapidly that the contour of the parts is not changed by the destructive process. The secretion is moderate, mucoid rather than purulent, and gray, thick, ropy, and tenacious. While the edges of the epiglottis may be eaten away here as elsewhere, there is along with the necrosis a thickening of the parts. Ankylosis of the crico-arytenoid articulation may occur; when this is the case it results in fixation of the cord in adduction. If the description of the syphilitic and lupous lesions is compared with that of the tubercular, the differences are obvious. Unless ulceration has taken place, perichondritis may be mistaken for it. In malignant disease the swellings are much more regular and nodular; the membrane is more inflammatory in appearance and the secretion profuse, purulent, and often sanguineous.

Prognosis.—As has been already stated, this is very grave, because it usually indicates a virulent general tuberculosis, and in ulcerated cases inanition results from the local interference with alimentation. The average duration of life is probably not more than two years. The writer's cases of tubercular ulceration which were under the beneficent influence of the Colorado climate show an average duration of three years and two months, while of the non-ulcerated cases it was six years. The writer found that in cases of phthisis treated by him in Colorado, of the non-laryngeal 71 per cent. improved, but of the laryngeal only 49 per cent. improved. Of these the non-ulcerated showed 68 per cent., and of the ulcerated only 25 per cent., improved. Taking the condition of the throat, without regard to the ultimate fate of the

patients, the results were much better, there being local permanent arrest of the disease in 64 per cent. It would therefore appear as if laryngeal tuberculosis, if it were not complicated by pulmonary tuberculosis, is as susceptible to treatment as the pulmonary form, though, in fact, it rarely is so, because in most cases there is the double disease to contend with. While there is in this, as in all other tuberculous infections, more or less inflammation, in the great majority of cases the inflammatory process is very feeble, anæmic, and often catarrhal in character.

Early recognition and appropriate climatic change, with local treatment, have a very marked influence on the prognosis, as has the tolerance of the throat to treatment and the state of the accompanying pulmonary tuberculosis. A few cases of spontaneous arrest of the tubercular ulceration are reported, but the disease, as a rule, shows but little natural tendency to repair.

TREATMENT.—It is only possible in these pages to discuss general principles. The climate best suited to the general tuberculous condition should be resided in, and this in the majority of cases is a high and dry one. As expressing the writer's views upon the local treatment, he will quote from a paper read by him before the Pan-American Congress in 1893:

"The first essential is the toilet of the throat—that is, the removal of mucus, pus, etc. and the washing of the membrane—and this is in most cases best procured, and with the pleasantest effects, by a free spraying with Dobell's solution. The old injunctions were to use sedatives and avoid stimulants for a tubercular larynx. Now, in the majority of cases, if the choice lay only between sedation and stimulation, I would say stimulate. While there are, often continuously, and generally at the onset, hyperæmia and hyperæsthesia of some of the parts, yet the underlying condition is an anæmic one, the congestions are chronic, not acute, and the real good of treatment almost invariably comes from stimulation, ranging from the mild stimulation of weak menthol and nitrate-of-silver solutions, etc. to the cauterization with lactic acid and the scraping of ulcerated surfaces with the single curette, or even the removal of tuberculous masses with the double curette of Heryng or Krause. Cocaine 10 per cent. to 20 per cent. should of course be used to avoid the pain of treatment, while for pain at other times a spray of antipyrine 10 per cent., will generally give more prolonged relief. I should extend this paper beyond its proper limit were I to enter further into the details of treatment, but may mention that astringents, iodoform, etc., are often of service. I believe in using the cotton applicator, the powder-blower, and the spray as the case demands; also I must mention the valuable aid I have derived from the Sass inhaler, particularly with the use of benzoin inhalations."[1]

℞. Tincturæ benzoini compositæ,	1 part;
Glycerini,	1 part;
Alcoholis,	1½ parts.

[1] *Therapeutic Gazette*, Nov. 15, 1893.

Lupus of the Larynx.

This is a rare disease. It usually extends from the skin to the respiratory tract, but there are a few instances where the larynx has been primarily attacked. The connection between lupus and tuberculosis is a close one, in that the tubercle bacilli are found in lupus as in other tuberculosis, and inoculation with lupous material has produced tuberculosis. It, however, differs from most other tuberculous affections, particularly the pulmonary form, in its remarkable chronicity and its local limitations. It is usually connected with manifestations of a strumous diathesis, and is not uncommon in the phthisical, although the connection is not clearly marked. Heredity does not appear to influence the disease directly. It is more common in males than in females, and the majority of cases develop between the ages of ten and thirty. The first stage is that of infiltration ; the second, of a characteristic nodular thickening, causing an irregular outline of the organ attacked. The tissue disappears under this process without evident ulceration and without self-proliferation, sloughing, or any secretion. The process is exceedingly slow, but in time the thickening becomes enormous.

Symptoms.—The onset is insidious. There is no pain and very little, if any, irritation, cough, or interference with deglutition, at least until the epiglottis is considerably involved. Dyspnœa is not present unless the thickening produces stenosis.

Diagnosis.—The epiglottis is usually the primary seat from which the disease extends to the ary-epiglottic folds, ventricular bands, the commissure, and hyaline cartilages, but perichondritis with necrosis never results. The disease causes loss of tissue without repair, and extensive distortion of the parts. Extending into the surrounding membrane are seen small, pale grayish, rounded nodules. It is without the hyperæmia, rapid destruction, and purulent secretion of syphilis, the membrane appearing pale and exsanguinated as in tuberculosis, but it can be distinguished from that disease by the excess of infiltration, the absence of pus, ulceration, or sensitiveness. The microscopical examination of a portion of the growth absolutely confirms the diagnosis.

Prognosis.—No case of spontaneous cure is recorded, but treatment has often arrested the disease in an early stage. Occasionally tracheotomy has to be resorted to on account of stenosis.

Treatment.—The local treatment for which most success is claimed is the application of nitrate of silver, 420 gr. to 1 oz. The use of chromic acid fused on the end of a small probe is also recommended. Scraping with a curette, followed by the rubbing in of lactic acid or the use of the galvano-cautery, is an excellent method. The general treatment should be similar to that for strumous cases. Cod-liver oil and tonics should be administered, and almost specific action is claimed for arsenic, especially in the form of arseniate of sodium.

NEW GROWTHS OF THE LARYNX.

Benign Tumors.

Bosworth considers that the importance of benign tumors has been somewhat exaggerated, as, although they may seriously impair phonation, they are not a grave menace to life. He states that they occur in the following order of frequency, viz.: papillomata, fibromata, cystomata, myxomata, adenomata, lipomata, angiomata, and enchondromata, and that the development, progress, and symptoms of these various forms are practically identical.

Etiology.—The causes are obscure. The disease has been ascribed to the following causes: a preceding catarrhal inflammation, over-use of the voice, eruptive fevers, the inhalation of irritative vapors, exposure to cold, and a papillomatous diathesis. They are apt to appear between the ages of forty and sixty, and more frequently among men than women.

Symptoms.—Their most common seat is upon the cords or adjacent membrane, interfering with their vibration. Dyspnœa is occasionally present, and when it exists is generally most marked during inspiration. The voice is usually weak and easily fatigued. Reflex disturbance and cough are not often present. When the growth is large there is a sense of discomfort and fulness, but not often pain. Attacks of dyspnœa at night may occur in young children from a resulting catarrhal laryngitis. The sputa may occasionally be tinged with blood from the erosions from friction upon the tumor.

Papillomata are the most common form of laryngeal tumors. The anterior portion of the vocal cords is their most frequent seat. Their development is slow in adults, but rapid in children, in whom they sometimes extend below the glottis—a rare condition in adults.

Fibromata are small, rounded, smooth, non-pedunculated tumors—are sometimes cystic; usually single, and generally develop on the cords. They are covered with a hyperæmic mucous membrane and are surrounded by a well-marked areola.

Cystomata are not common. There is usually a single tumor, light red in color, smooth, round, movable, and easily compressed. It is found most often on either the epiglottis or the cords.

Myxomata are rare. Their seat is always upon the cords. They are, as a rule, unilateral. They vary in appearance between that of myxomata elsewhere and that of papillomata. They may be pedunculated, multiple, or sessile.

Angiomata are usually found upon the vocal cords, and are almost invariably unilateral. They vary in size from that of a pea to that of a hazelnut, and occur in adult life.

Enchondromata usually grow upon the cartilages in the following order of frequency: cricoid, thyroid, epiglottis, and arytenoid. They appear in adults, and, growing slowly, develop a large, irregular, hyperæmic, sessile, immovable tumor, which, projecting into the laryngeal cavity, causes dyspnœa. They often develop erosions through friction.

Adenomata.—Bosworth thinks it doubtful if such growths have ever been found in the larynx.

Lipomata.—There is only one case reported where they did not grow from the ary-epiglottic folds and fall externally in the hyoid fossa.

Diagnosis of Laryngeal Tumors.—This can only be definitely made with the laryngoscope. The special form of the tumor must be judged from its usual characteristics, which are more fully discussed elsewhere.

Prognosis is favorable. The only danger, and that an exceptional one, is from their being of a size to interfere with respiration. Papilloma is the most rapid in development, especially in children, and is the only recurrent form. Malignant degeneration has been proved by Semon to be extremely rare.

Treatment.—The tumors can usually be removed through the larynx by the instrument appropriate to the form of growth. Forceps such as the Mackenzie are the most generally suitable. Sometimes the growth is best destroyed by cauterization. It is usually advisable to cauterize the seat of the tumor for a few weeks at weekly intervals. Occasionally the size of the growth makes an external operation necessary.

Malignant Tumors.

Sarcoma is rare and its causes unknown, though inflammatory conditions have preceded its development in many cases. It appears to be somewhat more frequent in men than in women, and generally occurs between the ages of fifty and seventy. It may appear in any part of the larynx, but is most frequently found upon the cords. It occasionally develops in the trachea, and it is usually unilateral. Hoarseness and aphonia, followed by dyspnœa and cough, are usually among the first symptoms. Dysphagia occurs when the epiglottis is involved. Lancinating pain radiating toward the ear is usually present. Erosion causing blood-stained sputa occurs early. The growth of the tumor is generally rapid, so that it may fill the larynx in three or four months. Cachexia is not marked until the later stages. The cervical glands are seldom involved, and the general extension of the disease is rare.

Diagnosis.—The tumor is semi-opaque, grayish or sometimes pinkish in color, rounded, irregular, and soft. Erosion is common, but ulceration rare. There is a muco-purulent, tenacious, ropy secretion. The microscope must be used to confirm the diagnosis.

Prognosis.—The disease is almost invariably fatal; recurrence is usual, and death generally occurs in less than twenty months.

Treatment.—Usually removal through the larynx affords the best chance of success. If this cannot be done, thyrotomy or resection of the larynx may be attempted.

Carcinoma.—Carcinoma of the larynx is exceedingly rare. Heredity has been found in 25 per cent. of the cases. It arises most often between the ages of fifty and sixty, and in men rather than in women. Laryngeal catarrh and over-use of the voice are possible causes.

Epithelioma.—Epithelioma is the most common form of cancer of the larynx. It may appear in any part, but most often upon the ventricular bands. It does not usually infect the cervical glands, unless

the disease is seated in the epiglottis, arytenoids, or ary-epiglottic folds. At first the symptoms resemble those of a benign tumor; respiratory disturbances next appear, and dysphagia and profuse salivation. If the disease affects the upper portion of the larynx, the tumor usually becomes excavated by extensive ulceration, with free bleeding as a result. The glands when involved are rapidly enlarged. At first there is a slight cough due to an excess of the healthy secretion, but an unhealthy, copious, thin, sero-, muco-purulent discharge ensues upon ulceration, with a characteristic musty fœtor to the breath. Hemorrhage is frequent and often serious.

Diagnosis.—An early diagnosis is not easy, as infiltration tends to spread deeply into the surrounding parts, with but little superficial swelling. Later, "an irregular, broadly infiltrating, ulcerated mass" distorts the parts with its peculiar fetid secretion. The rapid progress of the disease, the age of the patient, and occasionally the swelling of the cervical glands serve to confirm the diagnosis of sarcoma.

Prognosis.—Cancer of the larynx is peculiarly distressing and invariably fatal, but is rather slow, and the average duration of life is somewhat longer than in cancer in other parts of the body. The local application of antiseptics, such as electrozone, peroxide of hydrogen, or a ½ per cent. solution of pyoktanin assists disinfection, while europhen is best for diminishing the fœtor. Cocaine with antipyrine, applied in a spray or morphine powder blown on the surface, will relieve pain. Removal of as much of the growth as possible by operation, or if recurrence takes place total extirpation of the larynx, should be practised. Cauterization is to be condemned, as it serves only to stimulate the growth.

Prolapse of the Laryngeal Ventricles.

This accident has occasionally occurred. It appears to be due to violent coughing and is usually of gradual development. The symptoms that it causes are some loss of voice and dyspnœa.

Bosworth writes: "The tumor is a smooth, round, or spindle-shaped mass, pale pink or slightly injected, and lies upon the vocal cord, apparently emerging from the ventricular fissure. Its outline is regular, and it is soft and easily indented, which should serve to distinguish it from the hard, dense, irregularly nodulated, non-pedunculated fibroid tumors, which, morover, never spring from the laryngeal ventricle. The long duration of the disease and the absence of ulceration or glandular involvement will exclude malignant origin of the tumor."[1]

The dislocation cannot be reduced, and may need removal by the snare or by thyrotomy, but the swelling may sometimes be sufficiently diminished by the application of astringents.

Foreign Bodies in the Passages of the Larynx.

Liquids are usually expelled by coughing, unless they enter when the larynx is insensible, as during sleep, coma, or intoxication, when the symptoms would be severe dyspnœa and the presence of moist tracheal râles.

[1] *Twentieth Century Practice of Medicine*, vol. vi. p. 460.

Occasionally fistulous openings between the larynx and the œsophagus allow of the entrance of liquids.

The application of strong medicated solutions to the larynx sometimes causes spasm of the glottis.

Blood or pus may lodge in the larynx in finding its way up from the lower air passages. When coughing fails to dislodge the liquid the head and shoulders of the patient should be directed downward, the tongue drawn well forward, and artificial respiration used. Tracheotomy may have to be resorted to in cases of severe glottic spasm.

Solids.—The presence of a solid foreign body is often a serious accident. It occurs more often in children than in adults. An unlimited variety of foreign bodies which are small enough to be drawn into the larynx have been lodged in it. Food, teeth, pebbles, buttons, pins, and coins are perhaps the most common. The writer remembers a case in which he experienced some difficulty in removing an open safety-pin from the larynx of a child, and also in an adult a piece of hard lozenge was removed, which had been lodged in one of the ventricles for several weeks.

SYMPTOMS.—The primary symptoms are those of choking, spasm, coughing, and distress. Later there may be frothy and bloody sputum, with interference with the voice and respiration. These symptoms may intermit, in which case the object is usually a movable one. Where the foreign body has remained some time the symptoms may be mistaken for those of phthisis.

DIAGNOSIS.—The sudden onset of the symptoms, the often intermittent character of the dyspnœa or spasm, and the absence of fever serve to distinguish the presence of a foreign body from disease. A laryngoscopic examination should always be made. Pain is generally present and can usually be located.

TREATMENT.—The patient should be laid on his back and directed to take a slow, deep inspiration, and then to violently force out the air, the chest being struck at the same moment. Tickling the nasal cavity with a feather, and so causing the patient to sneeze, will sometimes dislodge the object. If these measures fail, the foreign body can sometimes be removed with the Cusco forceps. Occasionally, where the object is rough, it is best to crush it with the forceps before removal. In children an anæsthetic is frequently necessary. Should these measures not succeed and the symptoms be urgent, tracheotomy may have to be resorted to. In the case of a fish- or other small bone repeated applications of vinegar or hydrochloric acid (2 per cent.) on a cotton-wool tampon will soften the bone and render it easier of removal.

NEUROSES OF THE LARYNX.

The most satisfactory classification of the laryngeal neuroses is that based on their clinical manifestations, which Bosworth defines as follows:

"1. Sensory neuroses, under which head we consider hyperæsthesia, anæsthesia, paræsthesia, and general neuralgia;

"2. Paralysis involving the nerve trunk or nerve centre, as follows: Superior laryngeal paralysis, inferior or recurrent laryngeal paralysis,

bilateral paralysis of the abductors, and unilateral paralysis of the abductors;

"3. Paralysis of individual muscles; these are—Unilateral paralysis of tension, bilateral paralysis of tension, unilateral paralysis of the abductors, bilateral paralysis of the abductors, and paralysis of the arytenoideus muscle;

"4. Functional neuroses, the only affection coming under this head being hysterical aphonia;

"5. Spasm of the glottis: this disease differs in such marked manner, according as it develops in child life or in adult life, that it seems wiser that we should consider it under the two heads of spasm of the glottis in children and spasm of the glottis in adults;

"6. Inco-ordination of the laryngeal muscles, including chorea of the larynx, aphonia and dysphonia spastica, and laryngeal vertigo."[1]

Hyperæsthesia.—The reflex sensibility of the larynx differs greatly in individuals according to their temperament. It is more or less exaggerated in certain diseases, especially tuberculosis, and sometimes in carcinoma. It is usually quite marked in acute inflammations, and sometimes in certain chronic ones, notably in chronic alcoholism.

Anæsthesia is dependent upon some impairment of the action of the superior laryngeal nerve. It is not an infrequent sequela of diphtheria; it may appear in chronic inflammatory processes and in syphilis. It is generally found in connection with brain tumors, general paresis, and during the initial stages of affections of the medulla.

Paræsthesia.—Perverted sensations, such as a frequent inclination to swallow, an uncomfortable "cottony" feeling, as from the presence of a foreign body, are occasionally experienced. These symptoms are generally due to some lesion, such as hypertrophy of the pharyngeal lymphatics or one of the tonsils. In neurasthenics or in those in whom the general health is much depressed perverted sensations may be experienced in the larynx. The causes are imaginary or greatly exaggerated, but when the cause cannot be readily ascertained the other portions of the respiratory tract should be examined.

Neuralgia.—Neuralgia may occur as a result of anæmia, rheumatism, gout, malaria, or other general conditions. It is not often present in syphilis or lupus, but frequently occurs in phthisical and carcinomatous ulceration and in neurotic individuals. It sometimes appears to arise from causes more or less remote, such as acute naso-pharyngitis.

The TREATMENT consists of the removal of the causes and the use of such measures as are appropriate to a neuralgia.

PARALYSIS OF THE SUPERIOR LARYNGEAL NERVES.

These nerves, in addition to supplying sensation to the laryngeal membrane, also give motor innervation to the crico-thyroid muscle and partly to the arytenoideus. The result, however, of complete paralysis of the nerves is loss of sensation over the entire mucous membrane, with incomplete approximation of the arytenoid cartilages and imperfect tension of the cords. The voice is greatly weakened and sometimes entirely absent. It is not a common affection. It most often occurs as

[1] *Twentieth Century Practice of Medicine*, vol. vi. p. 425.

a sequela of diphtheria, when there is a loss of sensation accompanied by a weak voice. In these cases the muscles of deglutition are usually also involved. The writer has noted it in hysteria and once in the convalescence of typhoid fever.

DIAGNOSIS.—Upon this subject Bosworth writes as follows: "I know of no lesion or condition which will produce the somewhat curious glottis which is observed where both the superior laryngeal nerves are paralyzed, the interesting point being that the vocal processes and arytenoid cartilages are rotated inward in such a way that the vocal processes are the only points which are in contact during phonation, an elliptical opening being observed in front and a triangular opening behind, as seen in the illustration. If, on the other hand, we have a unilateral paralysis, this triangular opening between the vocal processes is not seen, the contraction of the arytenoid muscles being accomplished through the innervation of the nerve of the opposite side; hence the laryngoscope shows, then, merely a relaxed condition of the cord, extending from the vocal process to its anterior insertion—a condition as seen in paralysis of the crico-thyroid muscles. The impairment of sensation in the mucous membrane lining the larynx would indicate a lesion of the superior laryngeal nerve."[1] Recovery is usual.

FIG. 9.

Bilateral paralysis of the thyro-arytenoidei and of the arytenoideus (Browne).

TREATMENT.—Local faradization, friction, and massage, with the administration of strychnine, general tonics, and hygienic measures, should be adopted.

PARALYSIS OF THE RECURRENT LARYNGEAL NERVES.

The motor innervation to all the muscles of the larynx except the crico-thyroid is derived from these nerves: as a consequence there is complete motor paralysis of all the muscles on the side involved, as the crico-thyroid is prevented acting by this general paralysis.

ETIOLOGY.—Pressure upon the nerve trunk is the usual cause, and this pressure is often due to aneurysm of the right subclavian artery, but has been observed as a consequence of pressure from a pleuritic adhesion at the apex of the lung or to a general pleuritic effusion, or some central lesion may give rise to it. It may follow diphtheria or occur in the course of acute laryngitis. Sometimes the cause is obscure. It is more frequent on the left than on the right side. Double paralysis is rare.

SYMPTOMS.—The voice is weak and low, and speech tedious. There is often improvement after a few weeks, for the reason that in unilateral paralysis the sound cord becomes trained, so that it approximates better to the one which is paralyzed. When the paralysis is bilateral the cords are too widely spread to allow of any vibration, and therefore there is complete aphonia. When cough, dyspnœa, etc. are present they are due to the causative disease.

[1] *Twentieth Century Practice of Medicine*, vol. vi. p. 429.

DIAGNOSIS.—In unilateral paralysis during adduction the healthy cord crosses beyond the median line, with its arytenoid cartilage passing a little in front of that of the paralyzed side. The obliquity of the rima glottidis is deflected from before backward toward the affected side (see Fig. 10). To observe this condition the mirror must be held in a position whereby the centre of the crest of the epiglottis is seen in a direct line with the centre of the arytenoid commissure during inspiration, and the patient directed to speak, when the narrowed chink of the glottis will appear in an oblique line, while the paralyzed cord remains motionless in the cadaveric position. An epiglottis which is naturally oblique may give the appearance of a recurrent paralysis. Under these circumstances the mirror must be held in the median line of the fauces, so that it aligns upon the soft palate and the pharynx.

FIG. 10.

Unilateral paralysis of left adductor. Appearance in phonation: the right cord is seen to come beyond the median line, while the left is found in the cadaveric position (Browne).

Bilateral paralysis is revealed by the laryngoscope, with the cords in a position between adduction and extreme abduction, where—as in an adductor paralysis, for which they might be mistaken—the cords are so widely spread as to be almost hidden by the lateral laryngeal walls, and the outward direction of the vocal processes gives to them a somewhat concave outline. To ascertain the cause the neck, chest, and nervous system may require close examination.

PROGNOSIS depends necessarily upon the causes, also upon the duration of the paralysis, recovery being rare after nine months, owing to the muscular atrophy that results.

TREATMENT.—This depends upon the cause of the disease. Little can be done for those in whom an incurable disease is the cause; but sometimes the pressure of the tumor may be relieved by stimulation of the nerve trunk with electricity, one electrode being applied to the nape of the neck, and the other in front over the larynx or introduced into its cavity, so that the paralyzed muscles may be stimulated, cocaine being first applied. The faradic is usually of greater benefit than the continued current. Electricity, however, often fails to be of service and may even prove irritating. Tonics, and especially strychnine, are called for when the affection has resulted from an attack of diphtheria or one of the exanthemata.

BILATERAL PARALYSIS OF THE ABDUCTOR MUSCLES.

The opening of the glottis is due entirely to the action of the superior crico-arytenoid muscles, and their innervation is derived from a distinct nerve centre in the medulla.

ETIOLOGY.—The causes of this paralysis are obscure and still a matter of dispute among the authorities. It is probable that at least the most common cause is some degeneration in the special nerve centres located in the medulla. Atrophy of the crico-arytenoid muscles occurs as a result of the paralysis, and is usually complete in eight months.

SYMPTOMS.—The chief symptoms are spasmodic dyspnœa during inspiration. Neither the voice nor the act of expiration is usually affected. The spasms are mild at the onset, but gradually become serious, recurring two or three times a day.

"A transverse section of the larynx, as seen in Fig. 11, shows that the upper surfaces of the vocal cords and adjacent tissues are hollowed in such a way as to present a valve-like orifice when closely approximated, something like that of the semilunar valves of the aorta. When the air is inspired it has a tendency to render the closure more complete, and this, I think, will in part explain the dyspnœic attacks. Aside from these attacks, the other symptoms will be those of the disease occasioning the laryngeal paralysis, such as a central nerve lesion, aneurysm of the aorta, tumor of the mediastinum, bronchocele, enlarged lymphatic glands, locomotor ataxia, etc."[1]

FIG. 11.

Bilateral paralysis of abductors (crico-arytenoidei postici). Appearance with deep inspiratory effort (Browne).

DIAGNOSIS.—During inspiration the cords are parallel, and the opening between them is not more than one-eighth of an inch. They act normally during phonation. The most important matter in the diagnosis is to discover the real cause of the paralysis.

PROGNOSIS.—When the disease has existed for nine months the normal action of the muscles is seldom restored, but the patient may live without improvement in his paralysis for many years. When the danger from suffocation is imminent tracheotomy can be performed and a tube worn permanently.

TREATMENT.—Local treatment should only be used if the cause is peripheral, when electricity, massage, and strychnine should be resorted to. Antisyphilitic treatment has often proved successful. Tracheotomy is not only of service for the relief of dyspnœa, but also generally makes local treatment more effective.

UNILATERAL PARALYSIS OF THE ABDUCTORS.

This occasionally arises from acute processes in the larynx. "Gout, lead-poisoning, rheumatism, diphtheria, typhoid fever, and the exanthemata have all been known to cause it."[2]

The SYMPTOMS are so slight that the disease is often overlooked, though sometimes it changes into the bilateral form. A laryngoscopic image shows the normal cord fully abducted, while the paralyzed one remains motionless, whereas in adductor paralysis the arytenoid cartilage of the healthy side overlaps its fellow. During phonation the appearance is normal. In this affection tracheotomy is not needed; otherwise the treatment is similar to that for the bilateral form.

PARALYSIS OF INDIVIDUAL MUSCLES.

These are affections where some change has taken place in an individual muscle. They are usually the result of over-use or over-strain

[1] *Twentieth Century Practice of Medicine*, vol. vi. p. 438. [2] Bosworth.

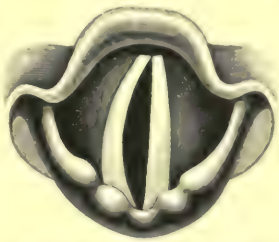
of the voice, or of the localized inflammation which may have arisen in the course of gout, rheumatism, malaria, or anæmia. Except when the arytenoideus is affected they are not of much importance nor are they common.

Unilateral Adductor Paralysis.—This is a very rare affection, but is stated to have ensued after diphtheria, exposure to cold, lead-poisoning, etc. More or less aphonia is the only symptom. During phonation the cords occupy the same position as in recurrent laryngeal paralysis, but their tension is not interfered with as in simple abductor paralysis.

Bilateral Adductor Paralysis.—The 5 cases reported by Mackenzie of this disease showed some movement. For this reason they were probably hysterical.

Paralysis of the Internal Tensors.—This affection, which is due to a paralysis of the thyro-arytenoideus muscle, is quite frequent and readily diagnosed. Its common cause is over-use or over-strain of the voice or chronic laryngitis. Phonation is impaired by reason of the voice being weak and its range limited. Both sides are generally affected. Inspection of a case of unilateral paralysis shows a semielliptical opening of the glottis, which is due to the issuing current of air causing an upward inflation of the cord. In the bilateral form there is seen a full ellipse. When the crico-thyroid muscle is involved the ellipse extends only from the vocal process to the receding angle of the thyroid. In paralysis of the thyro-arytenoideus muscle the upper surface of the vocal cord no longer remains broad and flat, but appears narrowed and cord-like.

Fig. 12.



Paralysis of left internal tensor (Bosworth).

Paralysis of the Arytenoideus Muscle.—This muscle is quite frequently affected in chronic catarrhal processes of the larynx. Diphtheria, hysteria, and phthisis are also causes. The paralysis prevents a complete approximation of the arytenoids, so that a wide triangular opening is left between the extremities of the vocal processes and the arytenoid cartilages; thus air escapes during phonation, and consequently the voice is more or less seriously impaired. Bilateral paralysis of the recurrent nerve may cause a similar condition, but in this case there would be an elliptical opening of the glottis extending from the vocal process to the thyroid angle in consequence of the paralysis of the crico-thyroid muscles.

Fig. 13.

Paralysis of the arytenoideus (Browne).

Prognosis.—When the condition has existed for some time the voice is not often restored, but there is quite frequently spontaneous recovery when it arises from diphtheria, from one of the exanthemata, or from exposure. On account of the inability to keep them at rest, paralysis of the internal tensors is the most difficult of

these affections to overcome. When myopathic paralysis occurs in the course of phthisis it is usually due to tuberculous infiltration, but may arise from simple anæmia and disappear with improvement of the general health.

TREATMENT.—Vocal rest is of the utmost importance. Local lesions must be treated. Electricity, especially with the faradic current applied directly to the muscle, is the most efficient remedy. The application should be made daily for ten to fifteen minutes at a time. Strychnine, iron, etc. and general hygienic measures should also be used.

HYSTERICAL APHONIA.

In this functional disorder there is imperfect muscular action and complete aphonia—a condition over which the patient has no direct control. It only affects the voluntary muscles and is always bilateral. It resembles the appearance of a bilateral paralysis of the recurrent nerve. It may be mistaken for a subacute or chronic laryngitis, but in this case the cords would be immovable. The symptoms might be mistaken for a case where closure of the cords was prevented by swelling in the commissure or over the arytenoid cartilages, but on inspection would show the absence of these conditions. It can be distinguished from bilateral paralysis of the recurrent laryngeal nerves, as in this affection abduction cannot take place, as it does in hysteria, by reason of its being an involuntary action. The onset is usually sudden. Cough exists—which is rare—in a genuine paralysis of the abductors. It occurs most frequently in unmarried women between the ages of fifteen and forty-five. Signs of an hysterical temperament can usually be found, and the diagnosis is made complete if the hysterical patient is placed under an anæsthetic, when she will talk during the second stage of anæsthesia.

TREATMENT.—It is always best to make use of local applications, giving assurance to the patient that a cure will be effected within some definite period. At the same time the extrinsic cause of her abnormal nervous condition must be sought for and remedied.

SPASM OF THE GLOTTIS (LARYNGISMUS STRIDULUS).

The disease is limited to purely neurotic cases. It differs so greatly in adults and in children that it must, therefore, be discussed under two heads.

Spasm of the Glottis in Children.—This is a reflex disturbance occurring in children of a neurotic tendency, and is most commonly due to some form of malnutrition. Rickety children are the most subject to this affection. Teething, indigestion, parasites, whooping cough, exposure to cold, acute catarrhs of the air passages, and pressure upon the laryngeal nerves by enlarged glands are causes. Irritation of the prepuce, cerebral or cerebro-spinal disease, may give rise to it. The affection most often occurs in male infants under two years of age.

SYMPTOMS.—The attack comes on suddenly without previous laryngeal symptoms. The child is usually seized at night, starting up from his sleep gasping for breath. The glottis may remain closed for as

much as twenty seconds. This is followed by several minutes of dyspnœa. The countenance is anxious and cyanotic. The spasms may occur several times, and then subside, to return the following night or remain absent for two or three days. These recurrences often continue for several weeks, when the child begins to slowly improve. A fatal termination from asphyxia is, however, quite frequent. During the attack general spasmodic movements are often seen. Attacks readily recur from slight exciting causes.

Diagnosis.—The age of the child, its neurotic tendencies, and the existence of some underlying cause, such as rachitis, assist the diagnosis. Bilateral paralysis of the abductor muscles, subglottic laryngitis, and laryngeal tumors may give rise to convulsive attacks of dyspnœa, but bilateral paralysis is a more chronic affection; the spasms are more prolonged, but much less severe, and are not accompanied by convulsive movements in other parts of the body. In subglottic laryngitis inspiratory dyspnœa often comes on at night, but there is a hoarse voice and a barking cough with expectoration and more or less pyrexia. Laryngeal tumors rarely cause spasms, and when they occur they are usually mild.

Prognosis.—A fatal result is not infrequent, particularly in the case of boys. The general strength of the patient and the severity and frequency of the attacks influence the prognosis.

Treatment.—The clothing round the neck must be immediately loosened and fresh air admitted to the room, while the child is placed in a semi-recumbent posture with his feet in a hot mustard bath, mustard plasters must be applied to the nape of the neck and cold compresses to the head. Subcutaneous injections of morphine and atropine should be promptly given in severe cases. The dose for a child of eighteen months is morphine gr. $\frac{1}{32}$, atropine $\frac{1}{500}$. When the symptoms are less urgent antispasmodics, such as musk, asafœtida, or castor, given by the bowel, are often serviceable. Chloral (gr. v–xv), dissolved in warm milk, given by the bowel, is sometimes better than morphine, though not so prompt. Leloir's method of compressing the phrenic nerve with the index finger placed between the two lower attachments of the sterno-cleido-mastoid muscle, repeated five times a minute, has been successful. Intubation or tracheotomy is sometimes necessary when the spasms are prolonged and there are general convulsions. Emetics, while of service in subglottic laryngitis, are useless in this affection. In the intervals between the attacks general hygienic measures should be taken and tonics, such as syrup of iodide of iron (♏v–xv), and an emulsion of hypophosphites with cod-liver oil, administered, while, of course, all possible causes of the attacks must be looked into and appropriate remedies applied.

Spasm of the Glottis in Adults.—Spasm of the glottis in adults is a reflex disturbance usually due to some underlying disease. It is never fatal. The entrance of food, drink, or a foreign body into the larynx, or the presence of a movable laryngeal tumor, is the most frequent cause. Interference with deglutition occurring in the course of syphilis or tuberculosis or from ulceration in the pharynx or the œsophagus, swelling in the fauces, or pharyngeal paralysis, may cause it. It is very rarely due to pressure on the laryngeal nerves or to a central lesion.

Symptoms.—The attacks are generally nocturnal, as in children, but

they are not otherwise similar in character, recurrences being neither frequent nor periodical.

Diagnosis.—The clinical history generally establishes the diagnosis. The absence of central nervous disease or of pressure upon the nerve trunks proves the reflex character of the affection, which is further confirmed if disease in the upper air tract is found. There are occasionally laryngeal spasms in early cases of tabes, but in this disease other signs of ataxia would be present. In the course of bilateral paralysis of the abductors symptoms of laryngeal spasm occur, but the laryngoscope would show paralysis of abduction, which would be absent in this affection.

Prognosis.—This disease very exceptionally requires tracheotomy to be performed, but when the case is reflex it is not fatal, and is usually readily susceptible to prompt treatment.

Treatment.—Local disorders must be attended to, and the hyperæsthesia of the larynx relieved by a spray of antipyrine, 10 per cent. cocaine 4 per cent., p. r. n. To allay the general nervous irritability antispasmodics, such as asafœtida, valerian, or bromides, should be used, accompanied with such tonic or hygienic measures as the general health of the patient demands.

Laryngeal Inco-ordinations.

Imperfect co-ordination of the laryngeal muscles may cause expiratory dyspnœa from spasm of the glottis. These disorders arrange themselves under three heads: (1) Chorea of the larynx; (2) Dysphonia spastica; and (3) Laryngeal vertigo.

Chorea of the Larynx.—In this affection the glottis is suddenly, momentarily, and completely closed during expiration, the air escaping with a dry, persistent cough resembling the barking of a dog, recurring every few minutes. The voice is not affected. Girls at the age of puberty are more subject to it than boys. The cords are driven together during the paroxysms "as if by some great external force, and after one or two seconds they are driven back to the sides of the larynx by a similarly quick movement" (Lefferts). With the explosive opening of the glottis comes the peculiar barking cough. Choreic movements in other parts of the body sometimes occur.

Dysphonia Spastica.—In this form the spasm of the glottis also occurs during expiration, but only when the voice is used. Aphonia more or less complete is the first symptom. A little later the glottic spasm occurs whenever the patient attempts to speak. The glottis is not always completely closed, but in this case the tone of the voice is that of a falsetto, owing to the extreme tension. Repeated efforts to phonate sometimes result in cyanosis. "By means of the laryngoscope we find normal vigorous movements of the larynx. An attempt to phonate will cause adduction of the cords in a perfectly natural manner, but the moment they approximate they contract spasmodically and close the phonatory glottis; one cord may overlap the other, and one arytenoid cartilage fall in front of its fellow" (Bosworth).

Laryngeal Vertigo.—In this rare disease there is immediate closure of the glottis, preventing expiration, followed at once by vertigo and

loss of consciousness. The patient may to all appearances be in perfect health when he coughs as the result of a slight tickling in the larynx, and this is immediately followed by the attack, which lasts a few minutes, and he recovers without any apparent bad results. These attacks in mild cases are limited to slight dizziness, with some dimness of sight. A catarrhal laryngitis sometimes exists, but the larynx is usually normal. The affection is unmistakable. It is not fatal.

TREATMENT.—In all these conditions the neurosis must be combated with general tonics, such as quinine, iron, arsenic, etc. Strychnine is usually too stimulating. Cold bathing, particularly in the form of showers and douches, is of great service. Any local lesion of the air passages must be removed. In chorea the exhibition of bromides and the use of the faradic current have been of service. In dysphonia spastica the voice must be used as little as possible, and the continuous current applied daily, the positive pole being placed over the cervical vertebræ, while the negative pole is moved along the whole course of the spine. When vertigo occurs the bromides should be freely administered.

PHYSICAL SIGNS OF PULMONARY DISEASE.

By ELBRIDGE G. CUTLER, M. D.

In the examination of a person with reference to suspected pulmonary disease the procedure should follow a definite line. The usual custom is to begin with an inspection of the naked thorax. The points of special observation are the color and general characteristics of the skin, the shape of the thorax, and the respiratory movements; next follows palpation of the chest, afterward mensuration of the same, then percussion, and finally auscultation. It is by pursuing this methodical course of examination that the physical signs of pulmonary disease are brought out, and can be put together so as enable one to draw a more or less correct conclusion as to the diagnosis in the individual case.

Inspection.—The patient should, as a rule, be bared to the waist, and either recline on a bed or lounge of convenient height or should sit in a chair supported by a high back. The light (preferably daylight) should be arranged to fall evenly on the patient. The examiner should view the patient from different standpoints—from in front of him, behind him, and at his side. By this means differences of expansion are better detected and unevennesses of surface best seen. In the recumbent position the patient should lie straight, with the legs stretched out even and the arms at the sides. The head should be elevated by a single pillow, and the chest should be free from all clothing. In the sitting posture the arms rest in the lap or the hands are clasped over the head, while the trunk rests squarely in the chair; the shoulders are held even and rest against the back of the chair.

The color of the skin is first observed. When it varies from the normal it is either pale, red, blue, yellow, yellowish brown, or otherwise pigmented. There are natural differences, varying with the race or family, age, climate, mode of life, so that no universal standard can be designated as the normal type for all mankind. One must judge from the appearance how the individual deviates from his normal.

(1) *Pallor* is a temporary result of faintness, fright, pain, chill, cold, and of a series of nervous influences which cause an anæmia of the cutaneous capillaries through cardiac paralysis or arterial spasm. It is more permanent when it is the result of loss of blood, loss of serum or other juices, or deep-seated disturbance of nutrition. The more profound such nutritive disturbance is, the greater is the accompanying atrophy of the subcutaneous fat tissues, consequent increased wrinkling of the skin, scaling off of the epidermis, atrophy of the skin itself, and therefore pronounced transparency of the cutaneous veins. In poor-blooded persons with rapid loss of tissue the skin has a sallow look,

with a yellowish or grayish cast, while the conjunctiva has a bluish look. Besides being pale, the skin may also be dropsical, so that it becomes stretched and is smooth, transparent, and waxlike. This is more especially the case where the blood is poor in plastic constituents.

(2) *Redness of the skin* results from temporary neuro-paralytic dilatation of the arteries due to mental causes, the various emotions, great muscular exertion, the taking of alcoholic drinks, a hot bath, fever, and the like. Occasionally there is a yellowish or greenish tint in a patient suffering from croupous pneumonia.

(3) *Blueness*, or *cyanosis*, is present when the visible skin and mucous membranes, instead of having the pale red or pink color of health, look bluish red. The intensity of this color varies. In the slightest degrees it is confined to those parts of the skin where the epidermis is thin and the cutis is especially rich in bloodvessels, as the lips, cheeks, conjunctiva, ears, tip of the nose, elbows, fingers, toes, cap of the knee, etc. If the cyanosis is very pronounced, it spreads over the entire skin and gives the patient a very peculiar appearance. The physiological cause of cyanosis is always the same: first, overloading of the blood with carbon dioxide; and second, a diminution or poverty of oxygen. The blood thus receives a venous character, which is shown by its abnormally dark color. To bring about this condition either the blood is too rich in carbon dioxide or too poor in oxygen; either the gaseous interchange between blood and atmospheric air inside the lung is lessened, so that the blood cannot give off enough carbon dioxide and change it for oxygen, or the rapidity of the blood stream within the smaller vessels is so much slowed that the blood takes up more carbon dioxide from the surrounding tissues than normal and at the same time gives up more oxygen. In cyanosis, then, there must be either a disturbance of the respiration or of the circulation. It is not essential that there should be disease of the respiratory or circulatory apparatus, or both, for the interference of the descent of the diaphragm in great gaseous distention of the abdomen may give rise to this condition; also paralysis of the diaphragm, paralysis of the vocal cords, especially the crico-arytenoid muscles, cause this condition. Catarrhal swelling of the mucous membrane of the trachea and bronchi, great collections of mucus in the same, fibrinous and diphtheritic exudations, cicatrices on the mucous membranes, muscular spasms, aspirated foreign bodies, compression by tumors (goitres, aneurysms, lymph gland, and other tumors) may cause it. Certain diseases may diminish the air space deeper in the lung, as cavities, miliary tuberculosis, so that under some circumstances cyanosis may prove a diagnostic mark. Frequently pressure from outside the lung may cause a diminution of the respiratory surface, as fluid or gas in the pleura or pericardium or tumors in the mediastinum. Diseases and disturbances of the circulatory apparatus are left out of account here, except in connection with the lung and its neighborhood; so also abdominal disease. Marked cyanosis may come from poisoning with various substances, as nitro-benzol, some of the modern antipyretics, etc. In certain cases of pneumonia the same is seen in slight degree.

(4) *Icterus* is at times seen in pneumonia, the so-called bilious pneumonia. It is usually a complication of duodenal catarrh, and is almost

always of slight degree. Formerly it was taught that it was seen most frequently in pneumonia of the right lower lobe, and was variously explained—for example, as dependent on the liver being pushed down by the solidified lung and its axis distorted, thereby causing hepatic venous congestion.

(5) *Local pigmentations* are frequently seen, resulting from the application of vesicants, sinapisms, salves, liniments, or they may be the remains of healed exanthems, ulcers of the skin, etc.

(6) *Pityriasis versicolor* is often found on the front of the chest and on the back over the shoulders in phthisis pulmonalis. It is, however, by no means rare in persons in perfect health, and has no special significance.

(7) A dirty, *yellowish color* is also frequently observed on the face in phthisis pulmonalis, quite distinct from pityriasis versicolor.

The muscular development should be noted, also the facies, the general position assumed by the patient, and the amount of subcutaneous fat tissue.

Œdema is seen chiefly in wasting, long-continued cases of disease of the lungs, and is usually confined to the extremities, chiefly the lower ones, though at times also present in the hands. Dropsy of one or both upper extremities occurs rarely, and is caused by compression of the axillary or subclavian vein by enlarged glands. More uncommon still is œdema of the entire upper half the body from thrombosis or compression of the superior vena cava by intra-thoracic tumors, exudations, and aortic aneurysms.

Emphysema of the skin is very rarely observed. It gives a crackling under the fingers like paper, or like the sound and feeling of lung containing air when squeezed. The skin is yellow, and pressure with the fingers leaves an indentation which rapidly disappears because the elasticity of the skin is not impaired. It appears sometimes over small areas, sometimes over large areas or even the entire body. Emphysema of the tissues of the neck follows perforations of the larynx and trachea due to ulceration. Emphysema of the tissues of the chest follows injury to the costal pleura and surface of the lung by a stab, shot, broken rib, and in rare cases it results from perforation of an abscess of the lung. Ruptures of the pulmonary alveoli may occur without any external violence, resulting simply from excessive over-distention, and lead to emphysema of the skin, the gas first escaping into the interlobular septa, thence into the mediastinum, and thence into the tissues of the neck; in these cases air first appears in the jugular fossa, then in the connective tissue of the sides of the neck, and lastly the chest.

The *size of the chest* varies in circumference, length, breadth, and depth. For convenience the chest is divided into front, back, and sides. The front is further subdivided into supra-clavicular, clavicular, infra-clavicular, mammary, infra-mammary, and supra, upper, and lower sternal regions. The back is subdivided into scapular, infra- and inter-scapular regions; the sides into axillary and infra-axillary regions. Vertical lines again are used, as the sternal, para-sternal, mammillary, anterior, middle, and posterior axillary, angle of scapula, and vertebral lines.

The *supra-clavicular region* is triangular and is bounded by the clavicle below, inside by the outer border of the sterno-cleido-mastoid

muscle, and outside by the outer edge of the trapezius. It has been fully established that the highest point which the top of the lung reaches is three to five centimetres above the clavicle. The *clavicular region* covers the inner half of the clavicle. The *infra-clavicular region* is bounded by the clavicles above, the sternum within, a horizontal line crossing the chest at the level of the third rib below, and outside by a vertical line dropped from the acromion process. The *mammary region* is bounded by the infra-clavicular above, the sternum internally, the acromion line externally, and below by an oblique straight line drawn along the middle of the sixth costal cartilage to the acromion line. The *infra-mammary region* is the portion of chest below the mammary region. The *supra-sternal region* is between the upper end of the sternum below and the lower border of the cricoid cartilage above and the sterno-mastoid muscles at the sides. The *upper sternal region* extends from the upper border of the sternum above to the level of the third rib below. The *lower sternal region* comprises the rest of the sternum below the third rib. The *scapular region* is the part covered by the scapulæ. The *inter-scapular region* lies between the scapulæ. The *infra-scapular region* is the part below the angle of the scapula to the base of the chest. The *axillary region* extends down from the apex of the armpit to the level of the insertion of the pectoral muscles. The *infra-axillary region* is the part below the pectorals to the base of the chest.

The *sternal lines* are three in number. They run vertically through the middle and right and left borders of the sternum, and are therefore called *mid-sternal* and *right* and *left sternal* lines respectively.

The *para-sternal line* runs vertically halfway between the sternal and mammillary lines.

The *mammillary line* runs through the nipple in the male. In the female it descends from the middle of the clavicle perpendicularly downward.

The *anterior axillary line* runs vertically along the anterior border of the axilla.

The *axillary line* descends perpendicularly from the summit of the axilla downward.

The *posterior axillary line* runs along the posterior border of the axilla.

The *scapular line* goes through the apex of the scapula when the arms are hanging at the sides.

The *vertebral line* runs between the scapular line and the spinal column.

Shape of the Chest.—A well-formed thorax should be nearly symmetrical on both sides. The two halves should measure practically the same. The curve beginning under the clavicle should gradually increase to the level of the nipple, from which point it should gradually diminish or recede toward the lower ribs. The supra- and infra-clavicular spaces should be almost on a level with the clavicles. There should be no grooves or depressions above or below the clavicles. The sternum and vertebral column should run in a straight line, and the scapulæ be symmetrically placed. In a well-nourished person, with good muscular development, the ribs should not be visible in the upper two thirds of

the chest, though they are so at the sides below. The nipple in the male is in the fourth intercostal space; in the virgin female, on the fifth rib.

Physiological deviations from this type are the rule. Among the more common ones are—prominence of the clavicles and of the insertions of the ribs at the sternum, marked convexity of the second and third ribs near the sternum on one or both sides, prominence of the sternum at the junction of the manubrium and corpus sterni, depression in some portion of the chest wall (less frequently seen in the ribs than in the sternum), either throughout the whole length or especially the lower part of it.

Pathological deviations from the normal may be temporary or permanent, and may be grouped as follows: First, enlargement, increased prominence of one or both sides; second, narrowing, diminution, sinking in of the whole of one or both sides; third, partial sinking in of one or both sides.

Enlargement.—Increase in the size of one half of the chest may be confined to the lower portion or may be of greater extent, and comes from a moderate quantity of abnormal contents of the pleural sac, as fluid, gas, tumors, or, very rarely, from increase in volume of the lung through hepatization of its entire extent. The greatest enlargement of an entire half chest is found in very large pleuritic effusions. Increase in size of the lower portion, seen most distinctly in the back and sides, is found in medium-sized exudations. Very moderate amounts of fluid which collect in the lower portion of the pleural sac behind do not cause any enlargement of the thorax. Enlargement of the thorax from pleuritic effusions begins with obliteration of the intercostal spaces. Next follows pressure of the fluid on the chest. If the effusion is only of medium size, so that the upper parts of the lungs still contain air, the obliteration of the intercostal spaces is confined to the lower portion of the thorax. The spaces above the fluid remain intact and sink in. So long as the lung can retract and thus give space for the fluid, there is no great enlargement of the thorax; when, however, the collection is so great that it takes up all this space, then pressure on the inner wall of the thorax begins and descent of the diaphragm follows, with descent of the liver on the right or spleen on the left. The mediastinum bulges toward the other side, and the heart is displaced to the right or left according to the location of the effusion. The increase in size of the thorax is here both in its vertical and transverse diameters.

If a pleural exudation is absorbed before a very long time has elapsed, the side of the thorax affected returns to its normal dimensions, provided the lung recovers its elasticity and power of expansion.

Air in the pleural sac (pneumo-thorax) gives rise to the same changes of form in the thorax as liquid. Pneumo-thorax comes on suddenly, either from external trauma of the pleura, shot, stab, fracture of rib, or usually from rupture of a superficial cavity of the lung which connects with a large bronchus. In rare cases pneumo-thorax has also been observed after perforation of an empyema into a bronchus, perforation of emphysematous pulmonary alveoli, perforation of the œsophagus, and perforation of the stomach through the diaphragm into the pleura. The lung usually retracts completely with great rapidity, and the side affected

takes the shape of the chest at the end of a deep inspiration. An increase in size beyond this does not occur till an inflammatory effusion begins, which usually follows from the introduction of low organisms, causing a pyopneumo-thorax.

Mediastinal tumors may cause considerable enlargement of one side of the thorax and prominence of the sternum.

Enlargement of both sides may come from emphysema of both lungs. The thorax is enlarged in all diameters, and in marked cases forms the so-called barrel-shaped chest. The sternum and ribs are more convex, and the intercostal spaces wider than normal, but not effaced. This change of shape affects the upper and middle portions of the thorax especially. The lower portions frequently appear flattened. The extent of the emphysema and its locality, together with its duration, on the other hand, may be such as to cause little or no change of shape in the thorax. In young persons with elastic walls it is more apt to be marked than in old people. The cause of the enlargement is the loss of elasticity in the lung, and consequently of its power of traction on the chest.

Diminution in size, or contraction, of half the thorax or the greater part of it is seen most frequently after absorption of long-standing pleuritic effusions or external perforation of the same on the affected side. If the exudation is large and has existed a long time, the lung has been reduced to a very small size and become atelectatic. Perhaps the visceral pleura has been also thickened, so that on absorption or evacuation the lung no longer expands, the thoracic wall sinks in, and the diaphragm is arched up higher. The same thing occurs in purulent collections which have perforated or been evacuated. In extreme cases the thorax is diminished in all diameters on the affected side, especially in depth. The forward curve is less; the chest is flat, at times sunken in; the diaphragm arches up higher; the ribs approach nearer together; the shoulder droops, the nipple approaches the median line, and the shoulder blade is nearer the vertebræ; the anterior mediastinum is drawn toward the affected side; the vertebral column becomes arched with its convexity toward the healthy side; the back muscles and intercostals have become paretic and atrophied. Corresponding to the contraction of the side a displacement of organs has occurred. The liver or spleen stands higher; the heart is dislocated to the right or left as the case may be. The most marked deformities of this class are seen in children. On the other hand, some portions of the lung may still be capable of expansion in these cases of late absorption of exudation, so that little or no change occurs in corresponding portions of the chest. The changes described above occur only in certain regions where the lung remains atelectatic, and over these places the chest sinks in.

The third group of pathological forms of the thorax includes the local depressions over greater or lesser areas. They only differ in extent and degree from the ones already spoken of. They occur in all diseases of the lungs accompanied by a contraction, whatever the cause, since a contracted portion of lung takes up a smaller space, and the atmospheric pressure outside causes the chest to sink in to supply the deficiency. The most frequent cause of these limited depressions is caseous degeneration, and, since such processes occur most frequently in

the anterior and upper portions of the chest, localized depressions are found most commonly in the supra- and infra-clavicular regions, sometimes on one side, sometimes on the other. They occur in much less degree in the supra-spinous regions. They are rare on the back even in great contractions of lung, because such contractions are usually caused by bronchiectasis, which does not lead to emaciation, and the thick muscles of the back more readily conceal ordinary differences of level.

Respiratory Movements.—In the normal condition the enlargement of the thorax is caused by the contraction of the diaphragm and of the intercostals. In the female the scaleni act in addition as well. In the male the diaphragm is chiefly active in this increase in size. The contraction of the diaphragm causes the abdominal organs to push out the abdominal wall, hence giving rise to the term abdominal type of respiration, the lower part of the chest being chiefly enlarged. In the female the intercostals are chiefly active in the enlargement of the thorax, so that the upper part enlarges most. This is called the costal type of respiration. A mixture of the two types is frequently seen—costo-abdominal.

In inspiration the chest enlarges in all its diameters. The breadth and depth are increased by the movements of the ribs and sternum, and the length by the descent of the diaphragm. This latter is the greatest increase.

In expiration the chest becomes smaller in the normal condition through relaxation of the inspiratory muscles and the pulmonary elasticity. The ribs and sternum return to their natural position and the air escapes from the lung.

The respiratory excursions of the thorax in the normal condition are equal on the two sides. If they are not equal, even slight differences are readily perceived, and point to an obstruction of the respiration in that half of the chest in which the movement is lessened. There may be diminution in the amount of air, or even complete absence of air in one lung, as, for example, in pneumonic consolidation or as the result of compression of a lung by fluid or gas in the pleura. Slight differences of expansion are often best detected by viewing the patient from one side, as already described above. Frequently the difference in the amplitude of the excursions is best seen in the movements of the shoulder blades; thus in a patient with a large pleuritic affusion the scapula on the affected side is moved but very little during inspiration, while on the healthy side it is elevated and the lower angle moves forward. The same thing is true of pneumo-thorax when the lung is wholly collapsed. If there is an obstruction to the respiration in both lungs of nearly equal extent and degree, the movements of the thorax on both sides are slight, in spite of great inspiratory effort, as in double emphysema of the lungs. If the interference with the respiration is in the upper lobe of one or both lungs, as in phthisis pulmonum, the diminution of the respiratory movements is confined to the upper part of one or both sides of the thorax. In the latter case, when the disease is slightly marked it is not so evident as when confined to one side, as there is nothing to compare it with.

In some cases the upper part of one, more rarely of both sides, is

seen to retract on inspiration, while it expands on expiration. This occurs usually between the first and the third ribs, where a portion of lung is condensed and contains several cavities or one large one, the reason being that the condensed lung cannot follow the enlargement of the thorax on inspiration, being unable to expand, so that the atmospheric pressure causes the soft parts to sink in over the affected area.

Sinking in during inspiration occurs very frequently in the lower portions of the thorax just under the edge of the ribs and the epigastrium, sometimes with implication of the ensiform cartilage and the sternal insertions of the lower ribs. They arch out again with expiration. This phenomenon is seen in extreme degrees of vesicular emphysema of the lungs and in laryngeal stenosis.

If there is marked obstruction of the respiration in the upper lobes, the lower lobes act more vigorously, the diaphragm contracts powerfully, the lower parts of the chest become widely distended, while the respiratory movements of the upper thoracic region are less. Phthisical patients often exhibit this type of respiration. On the other hand, if the action of the diaphragm is impeded, as when pushed up by large quantities of fluid or tumors in the abdominal cavity, or when it is depressed by pleuritic effusions and pneumo-thorax, the lower part of the lungs takes little part in the respiration.

The number of respirations in the adult male is fourteen to eighteen a minute. In women and children it is a little more, and in newborn babes forty and upward. One respiration occurs to about every four or four and a half pulse beats. The position of the body has a little influence on the frequency of the respirations. Respirations are more frequent in standing and sitting than in lying down. When attention is directed to the respiration it almost always becomes disturbed in its rhythm, either slowed or quickened. It may be voluntarily altered by being slowed, increased, made deeper or more superficial, or be stopped even for as much as a minute. It is most quiet and regular when the person is asleep or the attention is distracted. When the respirations are deeper than natural or more frequent, or both deeper and more frequent, the condition is called dyspnœa.

Physiological increase in frequency of respirations occurs with all considerable bodily exercise, especially that which increases the cardiac activity. Convalescents from severe diseases breathe faster when they rise up in bed, because their cardiac activity is thereby increased. Mental effects also increase the respirations; so, too, attention of the patient directed to himself or knowledge that he is the subject of observation does the same thing. The ingestion of food likewise increases the frequency in proportion to the abundance of the meal.

Pathologically, the frequency of the respirations is increased by the following causes:

1. Pain in a part of the body which the respiratory act sets in motion, either thorax or abdomen. The patient breathes more frequently and more superficially, so as not to move the parts in question any more than possible. The beginning of pleurisy is an example of this, as also pain in the ribs or muscles of the thorax in acute muscular rheumatism, and the severe pain in the abdomen in diffuse peritonitis.

2. It is increased by fever of any considerable degree from any cause.

3. Diseases which disturb the gaseous interchange in the lungs, as œdema of the glottis, croup, diphtheria, or tumors in the larynx, tumors which compress the trachea outside, catarrhal swelling of the bronchial mucous membrane, croupous pneumonia, œdema of the lung, caseous processes, fluid, air, or tumors of the pleura.

In general, dyspnœa is proportional to the suddenness of the diminution of the respiratory surface. If a lung, for example, has become collapsed through a pneumo-thorax within a few hours, dyspnœa is of a very high grade. If total collapse of a lung follows a slowly increasing pleuritic effusion, dyspnœa is very much less. If in addition to the diminution of the respiratory surface there are also pain and fever, as in pneumonia with pleurisy, the frequency of the respiration rapidly increases. On the other hand, the frequency of respiration is much less in certain chronic condensations of lung, which are larger in area than in pneumonia because of the freedom from fever and pain.

The frequency of respiration is further increased by diseases of the heart and circulation, which will not be considered here. An increased frequency of respiration follows certain diseases of the abdominal organs, tumors, collections of fluids or gas, which encroach on the thoracic space. These are also left out of account.

Finally, there are certain cases of dyspnœa which occur without any evident reason. These cases may be divided into two groups—one in which there has been said to be a general narrowing of the bronchi, bronchial asthma; and the other which depends on the excited condition of the vagus nerve or the respiratory centre in the medulla.

Certain so-called subjective attacks of dyspnœa must also be mentioned where the patient complains of oppression for breath, especially hysterical persons, and where objectively there is not the slightest dyspnœa evident. Usually such feelings of lack of air are temporary and disappear after a few deep inspirations.

In general the depth of respiration is inversely proportional to its frequency. In severe acute diseases of the respiratory apparatus—for example, pneumonia—the frequency of respiration is very considerable. Its depth is not increased. In chronic diseases of the respiratory apparatus, however, which do not use up the power of the body, as, for example, emphysema of the lungs, the depth of the respirations is sometimes extremely increased; the frequency of them is not especially increased.

As accessory muscles of inspiration are to be mentioned the scaleni, the sterno-cleido-mastoids, the pectorals, the subclavians, the levators of the ribs, the serratus posticus superior, the levator anguli scapulæ, and trapezius. All the muscles here named are used in enlarging the thorax. Another group of muscles enlarges the openings through which the air enters, as the levator alæ nasi, the levators of the soft palate, and the sterno-hyoid, sterno-thyoid, and thyro-hyoid, and the omo-hyoid, and the posterior crico-arytenoids.

A special kind of difficult respiration is that observed in diseases of the heart and of the brain, so-called Cheyne-Stokes respiration. In this form the respiration ceases at regular intervals for about a quarter,

half, or even a whole minute. A superficial respiration begins, the next following is deeper, and the inspirations become deeper, and at last very deep, without increase of frequency. When they have reached their climax the respirations progressively become less deep, and finally are very superficial, and at last respiration stops. After a quarter, a half, or a whole minute this characteristic type of respiration is repeated in just the same way. In the most marked cases the increasing and diminishing series of inspirations requires thirty to fifty seconds, about thirty inspirations in this time, and the expirations practically take up just about as much time, so that the whole process of respiration is a minute to a minute and a half long. The Cheyne-Stokes inspiration is observed in different diseases of the brain in the neighborhood of the medulla, hemorrhages, tumors, exudations, œdemas, and in heart diseases; also in basilar meningitis. It is also claimed that slight degrees of this form of respiration may be observed in relatively healthy persons of advanced age during sleep, and in children who are sleeping heavily.

Palpation.—In palpation the palmar surfaces of the fingers and hands are laid on the sides, the front, and the back in different corresponding places during ordinary and forced inspiration and expiration. By this means slight differences of expansion are frequently more readily detected than by inspection. By palpation we determine the thoracic resistance, painful areas, fluctuation, tactile fremitus, pleural râles, rub or friction, and pulsation. A healthy chest is to a certain extent compressible antero-posteriorly; the sternum can be sprung in toward the vertebral column a little, and with the cessation of the pressure it comes back again. The two sides may be compressed in the same way, but in much less degree. This compressibility appears to depend chiefly on the elasticity of the costal cartilages. There are certain physiological differences in the resistance to pressure on the thorax, depending upon the age. The thorax of the child is most compressible, that of the aged person the least so, because of the frequent deposit of lime salts in the cartilages, and there are all the differences of gradation between the two.

An early increase in the resistance of the thorax appears pathologically in phthisis not infrequently, and in doubtful cases may be of service in the diagnosis. In emphysema the same condition of resistance may be seen. It is also observed after middle age in the irregular rachitic thorax.

Pain in the thorax may be found in some cases by carefully going over each of the ribs and intercostal spaces with the fingers and making decided pressure along their whole course. Painful areas may be marked out with a pencil on the chest. There may be fractures or inflammations of bones, neuralgia of the intercostal nerves, rheumatism of the chest muscles, abscess of the thoracic walls, neuritis and inflammation, or new growth of the pleura or lung, which gives rise to this pain on pressure. A differential diagnosis must be made in all cases. In inflamed and carious ribs the pain is usually confined to a single rib and a narrow space of the same. Pressure on the rib itself brings out the sensitiveness, while the intercostal space is little or not at all sensitive. Then, too, there are usually redness and swelling of the skin over the sensitive spot. In intercostal neuralgia the pain, as a rule,

is confined to a single interspace. It sometimes extends from the vertebræ to the sternum, but often definite points along the nerve are especially sensitive. These are the so-called Valleix's painful points. One of these points is close to the vertebral column where the affected nerve comes out of the vertebral canal, called the vertebral point. Another is in the middle of the course of the nerve, where the lateral perforating branch reaches the skin, the lateral point. A third is near the sternum, where the anterior perforating branch penetrates the muscles, the sternal point. The differential diagnosis is rendered easier when the pain occurs in attacks of intermitting paroxysms. Rheumatic affections of the chest muscles are characterized by frequent and rapid change of place of the pains, and tenderness when the muscles are pressed with the fingers. Abscess may be recognized by the swelling, redness, and later on, fluctuation.

Fluctuation is not often found in the thorax on account of the rigidity of the chest walls. It is only when the collection of fluid in the pleura is very large, the intercostal spaces wide and bulging, that fluctuation can be felt, and here only by observing the precaution of placing the fingers near each other when trying for it. Fluctuation may also be detected in an empyema which is about to break spontaneously on the outside. The skin is first swollen, becomes boggy, then it reddens over a variable area, and at last comes the feeling of fluctuation. At this time by careful manipulation and gradually increased pressure the fluid may be driven away from under the skin back through the perforation into the pleura, and straining or cough may bring it back. This serves to distinguish empyema from all extra-pleural collections of pus. The pus may cover the greater part of the back and one side of the chest in exceptional cases of abscesses outside the pleura ; peri-pleuritis, abscesses of the thoracic muscles and ribs, and burrowing spinal abscesses are those most frequently seen.

Thoracic or Vocal or Tactile Fremitus.—When the hand is laid upon the thorax of a person speaking out loud, with each word a peculiar rapid trembling or vibration is felt in the chest wall, beginning with the commencement of the sound or word and also ending exactly with it. This is called pectoral or vocal fremitus. The vibration is quite like the feel of a sounding-board in a violin during play on that instrument, or a tuning-fork when struck. The vibrations of the vocal cords are transmitted to the column of air in the trachea, bronchi, and alveoli, and in turn to the tracheal and thoracic walls. The vocal fremitus increases with the loudness of the tone—the deeper the tone the greater the fremitus. The fremitus is stronger on the right than on the left side. The vocal fremitus is stronger over the interspaces in all parts of the chest than over the ribs. The different parts of the thorax vary in their power of transmitting fremitus. The front surfaces have it most distinctly, the sides a little less so, while the backs give the weakest fremitus. To give it a little more exactly in regions: the vocal fremitus of the supra-clavicular regions is considerably weaker than of the parts below the clavicle. As one approaches the median line the effect of the tracheal fremitus is made apparent by an increase in the sensation. The fremitus on the clavicles is still feebler. It is greatest in the inner third, less in the middle third, and weakest in the outer

third. Below the clavicles it is distinctly felt wherever there is lung, and is the most marked in this locality. On the sternum it is weakest over the manubrium, stronger over the processus ensiforme, and strongest over the gladiolus. On the middle of the back it is strongest over the fifth and sixth spinous processes of the dorsal vertebræ, and diminishes in intensity up or down the back. It is most distinct in the interscapular space, next the supra-scapular, next the infra-scapular, and least of all parts of the chest over the shoulder blades. These shades of difference have little clinical value, as it is the coarser changes which one looks for, and it usually suffices to remember at the sick bed that the vocal fremitus on the right is a little stronger than on the left.

The recommendation recently comes from Vienna to use a thin glass flask to detect the vocal fremitus in the following way: The bulb of the flask is grasped in the hand, and its mouth is placed firmly on the patient's thorax at different points while he articulates in the usual manner. The differences in pathological conditions are said to be more marked than in the ordinary palpation.

The pathological changes of vocal fremitus are either a diminution or an increase of the same caused by diseases of the bronchi, lung tissue, pleura, or the chest wall.

Diseases of the bronchi may cause either a weakening or an increase of the vocal fremitus. All considerable stenosis of bronchi weakens the vocal fremitus by hindering the conduction of the sound waves, or, under certain circumstances, completely abolishing them. Sometimes there is a temporary obstruction, as from mucus, pus, or clots of blood which narrow or completely close the bronchial tubes. Severe coughing sometimes does not clear such a bronchus. Also fibrinous exudations from the bronchial mucous membrane, foreign bodies lodged in a bronchus (most frequently the right one, where the size is greater and the air current stronger), cicatricial strictures, compression of the bronchial tubes by aneurysm, mediastinal tumors, considerable pericardial exudation, may cause weakening or suppression of the vocal fremitus. If these changes above described are found, they are accompanied by inspiratory retraction of the chest and a diminished participation in the respiratory movements of the affected side. In new growths in the lung tissue one meets the same weakening of fremitus quite frequently. The growth of a tumor into a bronchus may at times cause closure of a large air passage and thus bring about disappearance of the vocal fremitus. Similar conditions are developed in very extensive acute pneumonia. The only disease of the bronchi which causes increase of vocal fremitus is bronchial dilatation, but it only leads to this result when it is on the surface and lies immediately under the chest wall. If the dilatation is deep in and covered by aërated lung, there is no increase of the fremitus. Among the diseases of the lung which cause increase of vocal fremitus are all cavities and all conditions in which a great number of lung alveoli have become empty of air, so that the affected portion of lung approaches the character of a solid body. There are two conditions which must be fulfilled here: the focus of disease must lie on the surface of the lung, otherwise its influence is concealed by surrounding aërated lung; and, secondly, the bronchus leading to the focus of disease must be perfectly free or open.

If the alveoli of the lungs are filled with fibrinous masses, as in acute fibrinous pneumonia, or with cheesy masses, or if there is a solid tumor or a connective tissue cicatrix, an increase in the vocal fremitus will also be observed. The same is the case also when a large section of lung alveoli has become carnified by compression. If the compression is caused by collections of gas or fluid in the pleura, then the vocal fremitus is diminished over the collection. Very great pericardial exudation may compress the lung and give increased vocal fremitus at the sides and the posterior sections of the lower lobe of the left lung. In distention of the abdomen by tumors, fluid, and meteorism we find frequently compression of the lower portions of the lungs with increase of the fremitus.

Change in the vocal fremitus does not always follow disease of the pleura. Wintrich has shown by experiment that inflammatory membranous exudations have no influence. Collections of fluid or gas in the pleura are indicated by weakening of the vocal fremitus. It must be observed, however, that in the regions where the vocal fremitus is diminished or stopped as the result of a pleuritic exudation there may be circumscribed spots in which it is preserved or even increased, as shown by Lépine. These conditions are observed where adhesions have formed between the visceral and parietal pleuræ. In collections of air in the pleura the same thing has been observed by Ferber. It is not always alone the fluid in the pleura interposed between the lung and the chest wall which causes the weakening of the vocal fremitus, but the retracted lung may also compress the bronchi, and the effusion press on the chest wall and diminish its vibrations.

In encapsulated pleuritic effusions, and especially in the cases where there are several collections, vocal fremitus may give results of great diagnostic value. By palpating in vertical lines all over the chest the size of these collections may be determined, and the information utilized when there is question of operative interference.

If collections of fluid in the pleural cavities have become absorbed, a marked diminution of the fremitus may remain behind for a long time. This may be due to thickened pleura, to shrinking of a growth of connective tissue compressing bronchi, or to a retracted thorax which presents unfavorable conditions for transmitting the sound waves.

Pleural friction is chiefly felt in inflammatory conditions where an exudation has occurred from either or both the pleural surfaces. It gives the sensation to the touch of the rubbing or the crunching of loose, moist snow, or like the bending of new sole leather between the fingers, or like the feel of two pieces of new leather being rubbed together. It is characteristic that the fremitus is never continuous and equally marked, but there are frequent interruptions. One may find it either during inspiration alone, less frequently during both inspiration and expiration, and rarest of all during expiration alone. It sometimes is only observed at the end of a deep inspiration. It frequently wholly disappears after a number of deep inspirations, but returns again after a variable period of rest. If pressure is made in the intercostal spaces, the pleural friction or fremitus may be increased. Pleural friction may be felt without there being any inflammatory condition. Tubercular nodules and cancer may give rise to it. In the larger bronchi

tough mucus may occasion sounds on breathing which simulate it very closely and cause errors of diagnosis, but very frequently an act of coughing may dislocate the secretion and the condition ceases.

PALPABLE RÂLES.—The presence of secretion in the air passages may give rise to a sense of purring or bubbling in the thorax, its intensity depending on the quantity and the viscidity of the secretion. These râles are felt more readily in thin and yielding chests, such as are seen in children. They are felt most frequently in the anterior upper surface of the thorax, but occasionally also over the back.

Over a cavity where there are air and movable fluid at the same time a splashing sensation may be felt on sudden and considerable movement. This may be appreciated physiologically over the healthy stomach when partly filled with air and fluid, and pathologically in rare cases of pyopneumo-thorax over the chest.

Fine râles may be felt in emphysema of the skin and in prolapse of the lung.

PULSATING MOVEMENT in the thorax may be felt when the parts of the lungs next the heart are changed to dense airless tissue, as in hepatization or cancer or in certain cases of empyema. The latter may be differentiated from a pulsating aneurysm by the following points: (1) The location of a pulsating empyema necessitatis is almost always on the left side, low down, while aneurysm is mostly to the right and high up in the chest. (2) The pulsating empyema may almost always be made to disappear by pressure, and by increased expiration to increase in size, whereas in aneurysm this is not the case. (3) In perforating empyema the extent of the dulness is far greater than the tumor. (4) In aneurysm usually murmurs may be heard with the circulation.

MENSURATION.—The three chief diameters are to be noted (most readily accomplished by means of chest callipers): the vertical diameter from above downward—that is, the depth of the chest; the antero-posterior diameter from the median line in front to the spinous processes of the vertebræ behind, the sterno-vertebral diameter; and the diameter from one side to the other, the costal diameter. The points chosen for taking the diameters of the thorax have usually been the highest point of the axillæ, the level of the nipples, and the line of junction of the ensiform cartilage with the body of the sternum. These are also the places for taking the circumference of the chest by means of the saddle-tape or cyrtometer.

The *cyrtometer* is used for the double purpose of measuring the circumference of the thorax and of presenting its outline, so that it may be traced on paper. The simplest and at the same time the most practical form of the instrument is that described by Thompson in Wood's *Handbook of the Medical Sciences*,[1] and consists of a narrow strip of some metal, as soft lead, one-quarter-inch pipe, or a flat strip of any flexible alloy of lead ore, which can be accurately moulded to the body, and which when removed will still retain the shape which has been imparted to it. The strip should be long enough to surround the chest, be cut into two parts of equal length, and be slipped into a section of rubber tubing of sufficient length. The tubing not only protects the

[1] Vol. ii. p. 352.

skin, but allows the sections of metal moulded to the contour of the body to be removed without change of shape.

Among other forms of the cyrtometer are—that known as Woillez's, made up of numerous jointed links rather firmly connected together, except in one place, so as to retain the shape imparted to them; the Demoney chest measure, like the measure for the head; and the Evans cyrtometer, which consists of an elliptical frame of brass bar metal twenty-one by fifteen inches. The frame hinges at one point, and opens at the other corresponding to its greatest diameter. At the point of opening a pin operates to make the instrument firm when closed. The frame is perforated horizontally by thirty-one converging brass rods on each side, which may be fixed at any point by small set-screws. To operate the instrument the brass converging rods are pushed out to their limit, the frame is opened, and the patient placed within, his antero-posterior diameter corresponding to the shortest diameter of the ellipse and the sixteenth rod from the opening and opposite the middle of the sternum. Then the converging rods are pushed down against the patient snugly and the set-screws are fixed.

PERCUSSION.—The object of percussion is to disclose the condition of the parts underneath by means of light blows on the surface. It is usually described as of two kinds—immediate and mediate. In immediate percussion the blows delivered from the wrist are struck on the surface of the body with the open hand or fingers. In mediate percussion the blows are received on some intervening substance placed firmly on the parts to be percussed. Either a disk of ivory or hard rubber, called a plexímeter, or the middle finger of one hand is used. This latter method is the one most commonly made use of, and most satisfactory. To obtain satisfactory results certain precautions are indispensable. The finger which is struck is pressed firmly on the part and kept immovable, and the striking finger or fingers of the opposite hand should deliver a perpendicular, quick blow. The blow should be delivered from the wrist alone, the forearm, elbow, and shoulder being kept immovable. On children the blow should be made with the middle finger solely. Care must be taken not to allow the percussing fingers to remain in contact with the one used as plexímeter any length of time, as the resonance is thereby diminished. The stroke of the hammer in a piano on its string may be regarded as the type of percussion. As a rule, forcible percussion is not desirable, since the object is to obtain the note of the part immediately underneath the finger, and forcible percussion sets distant parts in vibration as well, and thus tends to create confusion. Hammers and plexímeters have no advantages of consequence over the fingers in ordinary practice, and the latter possess the merit of having the same texture as the parts of the body percussed, and so do not change the resonance of the tissue, and they enable one, moreover, to appreciate the sense of resistance given by solid tissue. There is no standard of resonance to which all chests must conform. Each must furnish its own, so that symmetrical points must be struck on the two sides with the same force. Percussion should usually be practised in straight lines, the pulmonary boders being first mapped out, and then the thorax gone over in detail. Five or six even blows should be given in rapid succession, and not two or three.

Percussion is either non-resonant or resonant.

Non-resonance is called flatness, and denotes entire absence of air in the part struck.

Dulness signifies diminished resonance.

Resonance indicates the presence of air in the part, and has four elements which must be described: They are—(1) *intensity*, (2) *pitch*, (3) *quality*, and (4) *duration* of the sound.

(1) The *intensity* of the sound signifies its loudness, and varies with the force of the blow and the skill of the percussor. It further depends on the character and thickness of the chest wall and the amount of air inside the chest. The intensity is less over a chest covered with a thick layer of fat, over a large female breast, and over thick pectoral muscles. The thick muscles of the scapula also diminish the intensity. Muscles in a state of contraction diminish the intensity, and in percussing the supra-clavicular region one must see that the patient does not turn his head toward the opposite side, or when percussing over the front of the chest that he does not thrust the thorax forward, or support himself on the hands when the back is being examined. The greater the thickness of the tissue which contains air, the greater the intensity of the sound.

(It is advisable to use some kind of pencil to mark out borders, and a very good one is to be obtained at any cosmetic store, enclosed in a tin case and composed of grease and lampblack or vermilion.)

(2) The *pitch* varies in different healthy chests and in different parts of the same chest. It is spoken of as being either high or low, with various qualifying adjectives or adverbs. The pitch is relatively low over healthy lung, and is closely related to the quality of the resonance both in health and disease. Though by no means absolutely essential for the detection of differences of pitch, a musical ear is undoubtedly an advantage. The pitch rises with diminution in the intensity or change in the quality.

(3) The *quality* is that element of the sound which gives to it its peculiar character and enables one to distinguish it from any other sound. Thus it is the quality of the violin notes which distinguishes them from those of the flute. There are only two qualities which are recognized clinically—the *vesicular*, which is produced by percussion over healthy lung and which is low in pitch, and the *tympanitic*, produced over relatively large collections of air, as contained in the stomach or intestines, or in the pleural sac, and which is high in pitch. A purely tympanitic note is rarely met with in the healthy chest, but a mixture of the two has been termed by Flint vesiculo-tympanitic. Tympanitic resonance is heard in health over the trachea and primary bronchi, and temporarily over the chest of a crying child.

(4) The *duration* of the tone is closely related to the pitch, and may perhaps be more readily recognized by the average listener than the latter. The higher the pitch, the shorter the duration.

Vesicular resonance is best heard by percussing over the healthy chest in the right upper front. The resonance here is of variable intensity, low in pitch, vesicular in quality, and of relatively long duration. It is called the normal vesicular resonance. It differs slightly in different individuals in all its elements, but a little practice will soon enable

one to always recognize it. The tympanitic quality is best obtained by percussing over the intestine or stomach, and it should frequently be compared with the normal vesicular resonance to keep the standards fixed in the mind.

VARIATIONS IN NORMAL RESONANCE.—We are now ready to consider the variations which normal resonance presents in different regions of the chest.

Supra-clavicular region: the resonance in this region varies much in intensity in different persons. The vesicular quality is most marked in the central portions. Toward the sternal extremity it acquires a tympanitic quality from the proximity of the trachea; it becomes vesiculo-tympanitic and the pitch is raised.

Clavicular region: the central portion has the vesicular quality more or less marked, the intensity diminishing as we approach the acromial extremity. Near the sternum it is vesiculo-tympanitic from the presence of the trachea, with slight elevation of pitch.

Infra-clavicular region: the resonance here is next in intensity to the axillary and infra-scapular regions. It is low in pitch, long in duration, and vesicular in quality, becoming vesiculo-tympanitic near the sternum from the presence of the primary and secondary bronchi, with therefore a rise in pitch.

Scapular region: the resonance here is decidedly less intense than in the infra-clavicular region, owing to the increased thickness of the chest wall and the presence of the scapula and its muscles. The quality is vesicular and the pitch low.

Inter-scapular region: the thick muscles of the back diminish the intensity, and the trachea and bronchi in the upper part of this region cause the resonance to be slightly vesiculo-tympanitic.

Mammary region: on the right the lung ends at the sixth rib in the para-sternal and mammillary lines, so that the intensity diminishes to this point. Moreover, the upper border of the liver arches upward to a slight extent, perhaps to the fifth rib, a relatively thin layer of lung covering it. On the left the resonance is diminished within the precordial space. This space extends from the third rib above to the fifth intercostal space below, and from the sternum to the mammillary line or just inside it. In women the varying size of the breasts diminishes accordingly the resonance in this region.

Infra-mammary region: the liver on the right and the stomach and a portion of the liver on the left modify the resonance in this region. On the right it is easy to define the line of flatness where the liver begins, and also where the liver dulness commences about an inch or two above this. These lines are raised or depressed by a full expiration or inspiration, and are therefore variable in locality. On the left the liver flatness extends three inches from the median line, while beyond it comes the tympanitic resonance of the stomach.

Sternal region: the resonance is tympanitic in the upper sternal region down as far as the second rib, because of the trachea and its bifurcation. From the second rib to the lower part of the third the resonance is vesicular. Below this point the subjacent organs, heart and liver, dull the resonance considerably.

Infra-scapular region: the resonance is quite intense here, and the

vesicular quality is marked, extending down to the tenth or eleventh rib in the scapular and vertebral lines. On the left the presence of gas in the stomach may give a tympanitic quality to the resonance.

Lateral or axillary regions: the resonance is relatively more intense and vesicular in these regions than elsewhere in the chest. On the right side the hepatic flatness begins at the eighth rib in the axillary line. On the left side vesicular resonance ends at the eighth rib in the axillary line, but very often a tympanitic quality is given to the resonance two or three inches higher, from gaseous distention of the stomach.

It has already been stated that the normal vesicular resonance is not identical in all persons in intensity, pitch, quality, and duration, so that there is no fixed standard with which we can compare a particular case. The standard for each individual must therefore be ascertained by comparing his two sides—in other words, each person furnishes his own standard. For purposes of comparison certain regions are better than others, as showing less disparity on the two sides. They are the supra-clavicular, clavicular, infra-clavicular, axillary, scapular, and inter-scapular regions. It is to be remarked, however, that the resonance on the left in the infra-scapular region is somewhat more intense, more vesicular, and lower in pitch than is the resonance on the right. The age modifies the normal vesicular resonance. In childhood, where the costal cartilages are more elastic, it is more intense and lower in pitch than in old age, where the cartilages are rigid and the lung more or less atrophied.

Abnormal Percussion Sounds.—Thus far we have considered only percussion in healthy subjects, and it remains for us to take up the modifications of disease.

They are—absence of resonance or flatness, diminished resonance or dulness, tympanitic resonance, and three subdivisions of the latter—viz. vesiculo-tympanitic, amphoric resonance, and cracked-pot (cracked-metal) resonance.

Flatness, absence of resonance, means absence of air in the part percussed. The sound produced is like that heard when percussing over the thigh or over a large collection of fluid in the abdomen. There is no pitch or quality to the sound; it is dead or woodeny. Flatness is found in the following pathological conditions: when there is liquid in large quantity in the pleural sac or in pulmonary cavities; when a large amount of fluid fills the air vesicles; in complete solidification of a large part of a lung; and in tumors within the chest.

(1) Fluid in the pleural sac is found in pleurisy, with effusion, empyema, and hydro-thorax. The smallest amount of fluid which can be detected with certainty in the adult chest by percussion is said by Seitz to be 200 c. c.; by others it is said to be about 400 c. c.

(2) Fluid in pulmonary cavities may be either in the shape of abscesses of the lungs or purulent or other fluids in phthisical or gangrenous cavities.

(3) Liquid in the air vesicles may come from pulmonary œdema, or blood may be extravasated from a hemorrhagic infarct. Pus may infiltrate the lung more or less, coming either from a perforating empyema, from the liver, or elsewhere.

(4) Solidification of the lung occurs in croupous pneumonia from exudation into the pulmonary alveoli. It is also produced by condensation or compression of the lung, due to air or fluid in large quantity in the pleura or pressure outside from a tumor.

(5) Tumors within the chest are aneurysms, sarcoma, carcinoma, lymphoma of the mediastinum, hydatids of the mediastinum, pleura, or lung, and dermoid cysts of the mediastinum. They vary in size and may occupy space at the expense of the lung. Flatness may also be caused by the encroachment of enlarged or displaced abdominal organs on the thoracic space.

Diminished resonance, or *dulness*, is found where solids or fluids are pathologically increased at the expense of air in the chest, the amount of increase being insufficient to cause flatness. The same may be caused by collapse of pulmonary lobules. Dulness varies, and may be slight, moderate, considerable, or great according to the relative disproportion of solids or liquids over air in the chest. The intensity is diminished. The quality remains the same. The pitch is raised in proportion to the degree of dulness. The pathological conditions which give rise to this sound are mostly the same as those which occasion flatness when existing in greater degree. Dulness, therefore, is met with in a small pleuritic effusion, empyema, hydro-thorax, a limited pulmonary œdema, hemorrhagic infarct, or abscess of the lung. It is also found in solidification of a portion of lung, in croupous pneumonia, phthisis, retracted lung from any cause, and a small tumor within the chest. Dulness may also exist in the first stage of croupous pneumonia before solidification has occurred, and also after resolution and absorption in the same disease. It is also at times met with after the fluid has been removed in pleurisy.

Tympanitic Resonance.—Any resonance which is non-vesicular is tympanitic, but there may be a mixture of the two, giving rise to the vesiculo-tympanitic note already mentioned as being normal in certain portions of the chest. The intensity of tympanitic resonance is not constant. It may be great or slight, but the pitch is always higher than in normal vesicular resonance. Percussion over the larynx and trachea is tympanitic.

The abnormal conditions which produce tympanitic resonance over the chest are as follows:

(1) Air in the pleural sac, pneumo-thorax: the resonance in this condition is greater than in health, is frequently found over the whole or greater part of one side, and the pitch is always raised.

(2) Pulmonary cavities containing air, as in phthisis: the tympanitic resonance is here circumscribed, corresponding to the size of the cavity. To give rise to this sound the cavity must be at least the size of a dove's egg and lie near the surface of the lung. The pitch of the tympanitic resonance over a cavity communicating freely with a bronchus is higher when the mouth is open than when it is closed. It is still more lowered when one nostril is closed.

(3) Solidification of a part or the whole of the upper lobe of a lung gives a tympanitic note derived from the trachea or bronchi. This may be found in pneumonia or phthisis before the stage of excavation. Dilated bronchi with solidified lung surrounding them may occasion the same thing.

(4) Tympanitic resonance may be conducted from the stomach over a part or the whole of the left side of the chest, especially when the lung is solidified.

Vesiculo-tympanitic resonance is heard in health at the sternal extremity of the supra- and infra-clavicular and clavicular regions, and in the interscapular region, and over the healthy chest of a crying young child. It is due, as before explained, to a mixture of the normal vesicular resonance of the lung and the tympanitic resonance of the trachea. It is high in pitch in proportion to the absence of the vesicular and predominance of the tympanitic element in the resonance. It is also heard in the following abnormal conditions: over the dilated air vesicles in pulmonary emphysema, over the relaxed lung in a chest one-third, one-half, or more filled with fluid. It is also heard over the upper lobe when the lower lobe is solidified by pneumonia, or over the lower lobe when the upper lobe is in the same condition.

Amphoric resonance is a musical or metallic intonation, such as may be produced by striking an empty bottle, pitcher, cask, or large rubber ball. It has a musical, ringing note of a metallic character, with a resonance which approaches an actual echo, so that it is sometimes called "metallic echo." It differs from the simple tympanitic note in that it is of longer duration owing to this resonance or echo, and is of higher pitch. The characteristics of the sound may be very well studied by distending the cheeks with air and then snapping them with the finger. The condition demanded for its production is a large cavity filled with air having smooth walls, which may or may not communicate with the external air. In the chest the amphoric resonance is heard over large pulmonary cavities, and in cases where air has accumulated in the pleural sac. Wintrich found that to give the amphoric resonance the cavity must have a length of at least six centimetres in the direction in which percussion is made. It must be quite superficial, bounded by homogeneous walls, not subdivided by bands of tissue which have escaped ulceration. It must not contain too much fluid, and, furthermore, the chest wall must not be too resistant so as to weaken the vibrations. In cases where the cavity communicates freely with a large bronchus the intensity of the resonance is increased by opening the mouth. The amphoric resonance is heard in pneumo-thorax as soon as the air in the pleura has reached a certain degree of tension, not too great. If the tension is great in the chest wall, the metallic sound may not be heard at any distance away, and auscultation of the chest must be practised at the same time with the percussion to get it. The echo is here often best heard by having the assistant place a coin firmly on the chest and then tapping it several times gently with another.

The cracked-pot resonance may be imitated by clasping the hands together crosswise, so as to leave a hollow between the palms communicating by a narrow opening with the outside air, and then striking the back of one hand on the knee. A sound like the chinking of coins is heard from the escape of air under pressure through the narrow opening. The same sound may be obtained in health by percussing strongly the chest of a crying child, or if the pleximeter is not snugly applied to the chest, or if the chest be hairy and not previously wet, to cause the hair to

stick close to the skin. The cracked-pot sound occurs in the following pathological conditions:

It is found most often over pulmonary cavities of moderate size communicating freely with a medium-sized bronchus superficially located, with an elastic, thin chest wall. These conditions are found most often in the infra-clavicular region, and the sound is best brought out during expiration and with the mouth wide open. One or two strong blows elicit the sound better than several. At times it can be detected best by bringing the ear or end of the stethoscope near the open mouth of the patient. Repeated examination or a collection of mucus may cause the sound to disappear temporarily, but reinflation of the lungs or cough with expulsion of mucus brings it back again. The cracked-pot sound is sometimes heard in pleurisy, in the relaxed portions of lung just above the level of the fluid; also occasionally in pneumonia, in the relaxed lung immediately adjoining the solidified portions, or over the latter. In neither of these cases, pleurisy or pneumonia, is the intensity increased by opening the mouth. The sound may finally be met with in pulmonary or thoracic fistulæ from any cause.

Sense of Resistance on Percussion.—Before leaving the consideration of the pathological conditions which affect the percussion note a few words must be said concerning a sense of resistance to the fingers, which is appreciated in those conditions of the chest which are accompanied by an increase in solid or liquid contents at the expense of the normal amount of air. In percussing the chest of a young person the fingers readily detect a certain amount of elasticity due to the flexibility of the parts underneath. This is less marked in the adult and still less marked in the aged patient, due to the increasing rigidity of the ribs and cartilages as age advances. A further increase in the resistance is encountered in cases where the bones of the framework of the chest are large, where the intercostal spaces have become narrow, and where there is a considerable development of fat, as about the female breast. The pathological change which gives rise to increased resistance on percussion over the lung is impermeability of the same to air. This may be produced by infiltration or contraction of the lung or a portion of it by fluid and tumors in the pleural sac. The parts are thus rendered less capable of vibration when struck, and the resistance increases with the diminution of the amount of air in the lung. An œdematous lung is less resistant than a hepatized lung, and this in turn less so than a large pleuritic effusion, while a large tumor presents the greatest resistance to percussion of any known condition of lung or pleura. This increase in the sense of resistance is often of value in aiding to mark the transition from air-containing to solid organs, where the ear fails to detect nice shades of differences in sound.

PULMONARY BORDERS.—The only borders of the lungs which can be defined by percussion are the superior and inferior borders and that part of the left anterior border which lies across the cardiac area.

Superior Borders.—The apex of the lung rises above the clavicle from three to five centimetres. The superior border extends from the inner end of the clavicle at first upward along the posterior edge of the sterno-cleido-mastoid muscle, and then over the shoulder in a gentle sweep to the spinous processes of the seventh cervical vertebra. On the back

the border is concave upward. The distinction between the pulmonary resonance of the apex and the tympanitic resonance of the trachea in front can best be made out by light percussion and with the patient's mouth open. The importance of determining these boundaries may be noticed in phthisis when one apex is often considerably retracted.

Anterior Borders.—The anterior borders begin at the sterno-clavicular articulation and run downward and forward to the middle of the sternum opposite the middle of the second costal cartilage; thence they run downward together, separated only by the mediastinum, to the fourth rib. From this point they differ in the following particulars: the right anterior border continues down to the sixth rib in the median line. Owing to the resonance of the sternum and to the fact that we cannot distinguish the sound of the right from that of the left lung, it is impossible to outline those portions of the anterior borders which underlie the sternum. In percussing down the left sternal line we notice dulness of the resonance beginning at the third rib. This dulness is due to the underlying heart, and becomes flatness on reaching the fourth rib. This line of flatness extends a short distance outward along the fourth rib, and then turns perpendicularly downward across the fifth rib. At the sixth rib it turns again to the left and is lost in the lower border.

Inferior Borders.—On the left side the inferior border lies as follows: In the mammillary line it begins at the sixth rib; in the axillary line it is at the eighth rib; scapular line, at the tenth rib; vertebral line, at the eleventh rib. The inferior border of the right lung is as follows: median line, base of the xiphoid cartilage; parasternal and mammillary lines, on the sixth rib, sometimes nearer the upper edge, sometimes nearer the lower edge of the rib; axillary line, at the eighth rib (it may be found as high as the seventh intercostal space or as low as the eighth intercostal space); scapular line, at the tenth rib; vertebral line, at the eleventh rib. With the acts of respiration the lower borders of the lungs ascend and descend, so that their position is not fixed, and there is thus what is called an active mobility of the lung which varies from one to two centimetres in the parasternal line to three to four centimetres in the axillary line. There is furthermore a mobility called passive mobility, due to changes of position, as when the anterior becomes lower when the patient lies down than when he sits up, or the lower border descends in the part accessible to percussion when the person reclines on the side.

Before finishing the subject of percussion a few words must be said about auscultatory percussion, a method first advocated by Laennec in the case of pneumo-thorax and already described under amphoric resonance. Since his time the method has been extended to more carefully map out the outlines of the heart, liver, and spleen, and also the stomach and intestines, when distended with air. It has also been used to more accurately outline tumors and cavities under certain conditions. The method, however, requires much practice and the aid of an assistant, so that, as a rule, the ordinary percussion is found to suffice for most clinical purposes.

Auscultation of the Healthy Chest.

In auscultation of the chest we listen to the sounds produced by respiration, voice, and cough. There are two methods of auscultation, *immediate* and *mediate*.

In immediate auscultation the ear is placed directly on the chest with or without the interposition of a towel or portion of the clothing. It has the advantage that the sounds within the chest are heard more loudly, and that a larger area can be examined at once, corresponding to the size of the ear, than by the mediate method. It is of service, therefore, in very sick persons, when the patient can be held up but a very short time for an examination of the back, and in children, who are sometimes frightened by the sight of any instrument. The disadvantages are that it is often impossible to adequately reach certain portions of the chest, as the supra-clavicular regions, and the examiner's own hair or whiskers sometimes bring in adventitious sounds quite like some of those of disease. Furthermore, in the female it is unpleasant, on the score of delicacy, for both examiner and patient when auscultating the front of the chest over the breasts. Lastly, it is not possible with it often to accurately localize certain sounds.

In mediate auscultation a stethoscope is used, either a single straight instrument, or the binaural one, which finds most frequent use in this country and is more satisfactory. With it the sounds are clearly conducted to the ear, and fine shades of difference are accurately brought out and localized. It is well, however, to familiarize one's self with both methods. In using the stethoscope the instrument should be nicely applied to the chest, but not so firmly as to give rise to pain. The examiner should kneel or place himself in such a position as to keep the head erect or nearly so, by this means avoiding an over-distention of the head with blood, which diminishes the acuteness of hearing. No portion of the clothing should touch the stethoscope, as otherwise additional sounds are heard.

The respiratory act consists of an inspiration and an expiration. These have different sounds over the trachea and over the lung, and we must now study them in detail, bearing in mind that each has, as in percussion, four elements—intensity, pitch, quality, and duration.

The sounds produced in ordinary respiration are sometimes feeble, so that we wish to increase them by forced breathing. In such cases it is advisable to show the patient how to do it so as not to alter the rhythm.

Respiration in Health.—*Tracheal Respiration.*—The inspiration heard over the side of the larynx has various degrees of intensity: it is usually great, the pitch is high, the quality bronchial or tubular like air rushing through a tube, and the duration a little less than the act of inspiration.

The expiration is always heard with forced breathing. The intensity is greater than in inspiration, its pitch is higher, the quality is tubular, and the duration is as long as or longer than the inspiratory act.

The characteristics, then, of normal tracheal or laryngeal respiration (heard over the larynx, trachea, or back of the neck) are an inspiratory sound of variable intensity, high in pitch and tubular in quality; an expiratory sound of greater intensity, higher pitch, and a duration as long

as, or longer than, the inspiration; and, because the inspiratory sound does not last quite so long as the inspiratory act, there is a slight interval between the two sounds. This bronchial respiratory murmur is heard loudest over the narrowest portion of the canal—namely, the rima glottidis—and thence it is propagated downward into the trachea and bronchi with gradually diminishing intensity. At the bifurcation of the trachea, opposite the fourth dorsal vertebra, between the scapulæ, usually only bronchial expiration is heard on the right of the median line, as the right bronchus is not only larger than the left, but it approaches more nearly to the surface than its fellow. Bronchial respiration is not heard in the normal chest except in the regions mentioned. It should be carefully and repeatedly studied so as to be firmly fixed in the mind.

Normal Pulmonary Respiration.—*Inspiration.*—To obtain the most typical form of normal pulmonary respiration one must listen over a part far removed from the trachea and bronchi, as the axilla or lower part of the back. The pulmonary respiratory murmur is heard only during inspiration, as a rule, throughout its whole duration, unless the breathing be superficial, when it is audible only toward the end. The intensity depends on the thickness of the layer of lung examined, the force of the breathing, the thinness and elasticity of the chest wall, and varies greatly in different individuals. There is a difference of intensity in the two sides and in different portions of the same side. The intensity therefore varies. The pitch is notably low compared with the tracheal respiration. The quality is peculiar, soft and breezy, like the sighing of wind through the leaves of a tree, and is called vesicular.

Expiration is sometimes inaudible, especially in parts away from the large tubes; as a rule, it is heard to be much less intense than the inspiration, with a lower pitch, a quality neither vesicular nor tubular, but blowing (it may be imitated by exhaling with the mouth open), and a much shorter duration.

To recapitulate: The inspiratory sound varies in intensity, is low in pitch, vesicular in quality, and lasts from the beginning to the end of the respiratory act; the expiratory sound follows immediately, and is less intense than the inspiratory one, is lower in pitch, has a blowing quality and a shorter duration, averaging about one fifth as long.

The inspiratory murmur is louder in front than behind; it is less purely vesicular in the sub-clavicular region, on the right side than on the left, and the pitch is higher. This is more marked the nearer we approach the sternum over the site of the primary bronchi. It is to be explained by the presence of additional bronchial tubes on the right, not found on the left side in the upper lobe, as pointed out by Cary. The intensity of the murmur is considerable in the axillæ; it is less over the mammary regions, and least over the scapulæ, while the pitch and quality remain normal.

Normal Vocal Resonance.—*Tracheal Voice.*—When the stethoscope is placed over the side of the larynx or above the sternal notch and the patient is requested to count one, two, three, in a moderate voice, one perceives a strong resonance and a sense of concussion, together with a thrill or fremitus. The sound seems concentrated and near the ear, while at times the words are heard more or less distinctly.

The elements here are vocal resonance, nearness to the ear or concentration, thrill or fremitus, and transmission of speech. When the words are spoken in a whisper there is no shock or thrill, but an intense high-pitched blowing sound, as if air were blown into the stethoscope. The words are sometimes transmitted to the ear.

Thoracic Voice.—This should be studied both with the stethoscope and the unaided ear. The patient should count one, two, three, with moderate force, and the listener place the ear or stethoscope over the middle of the right front or back below the scapula. The intensity depends on the loudness and pitch of the voice, as well as on the thickness of the wall, and hence varies with different individuals. It is less in women than in men. There is no sense of concussion. The sound is distant and diffused, in contradistinction to that heard over the trachea, which is near the ear and concentrated. It is accompanied by a sense of thrill or fremitus. Words are not transmitted. The characteristics of normal vocal resonance are, then, a distant and diffused sound, of various degrees of intensity, accompanied by more or less thrill or fremitus.

On comparing the two sides the vocal resonance is always found to be greater on the right than on the left throughout its whole extent in health, though the degree of difference varies in different persons. In the right infra-clavicular region it is more intense, nearer the ear, less diffused, and of a slightly higher pitch than on the left, owing to the additional bronchi mentioned above. The same thing is true with all the other regions, though the differences, as a rule, are less marked.

The whisper when heard over the middle of the right front is in most people a feeble, low-pitched blowing sound, corresponding to the expiratory sound in forced breathing, as pointed out by Flint. It is frequently inaudible in the scapular, infra-scapular, mammary, and infra-mammary regions. Where present it is always louder on the right side.

RESPIRATION IN DISEASE.—The respiratory signs in disease may be grossly grouped into two classes—first, where abnormal modifications of normal respiratory sounds are found, and, second, where new or adventitious sounds are heard.

In the first group there are the following modifications: increased vesicular respiratory murmur, diminished vesicular murmur, suppressed respiratory sound, bronchial or tubular respiration, broncho-vesicular respiration, cavernous respiration, broncho-cavernous, vesiculo-cavernous, and amphoric respiration, shortened inspiration, prolonged expiration, interrupted inspiration.

Increased Vesicular Respiration.—The sound is here abnormally loud or intense, the other characteristics remaining normal, the pitch being low and the quality vesicular. It is called also supplementary or puerile respiration, the former because that portion of lung where it is heard is doing extra or supplementary work; puerile, because it resembles the respiratory murmur heard over the chest of a child. Such increase on supplementary respiration is heard on the healthy side when the respiration on the other side is interfered with by disease, as in a large pleuritic effusion, pneumonia of the whole or greater portion of the lung, obstruction of a primary bronchus, or pneumo-thorax. It is sometimes mistaken for bronchial or broncho-vesicular respiration—an

error which careful attention to the pitch and quality, and a comparison with the trachea and some other healthy portion of lung, ought to eliminate.

Diminished vesicular murmur is incident to a number of conditions which affect the intensity of the respiration. These may lie either in the chest wall, such as pain in muscles or partial paralysis of the same; pain in intercostal nerves, as in pleurodynia; intercostal neuralgia, pleurisy, and pneumonia, or disease of ribs, which act by causing diminished expansion, and thus shallow respiration; deficient contraction of the diaphragm from paralysis; abdominal growth or effusions which offer mechanical obstruction or inflammation, such as peritonitis, which hinders the descent of it through pain, or they may be in the air vesicles or air passages or outside the same. Thus, dilatation of the air cells as in emphysema, blocking of the same with blood or serum as in pulmonary hemorrhage or œdema, the presence of air, liquid, or thickened pleura between the chest wall and the lung, may all give rise to it. Swelling of the bronchial mucous membrane, especially in the smaller tubes, incomplete obstruction of the same from the presence of mucus, serum, blood, pus, or any other foreign body, may cause it. Pressure of any tumor on one or more bronchi may give rise to unilateral diminution of respiration in a portion or all of one lung, as in aneurysm, mediastinal or peribronchial growths. A tumor may press on the trachea, and by narrowing the passage cause diminution on both sides, or the same thing may result from a cancerous or cicatricial stricture or laryngeal exudations or growths. The pitch and quality in all these cases remain the same, the intensity of the respiratory murmur alone being affected according to the amount of obstruction. In some cases of phthisis there is in certain stages a diminished respiratory murmur at the apex due to obstruction of bronchi, deficient movement of the chest, and exudation into the alveoli.

Absence or suppression of the respiratory murmur occurs under very much the same conditions as in diminution, and, as they are merely further advanced conditions of the same thing, they need not be repeated.

Bronchial or tubular respiration is heard normally over the larynx and trachea or over the back of the neck. Its characteristics have already been considered, but may be recapitulated. They are—an inspiration of greater or less intensity, of a high pitch and tubular or blowing quality; an expiration of still greater intensity, higher pitch, longer duration, and the same blowing quality as the inspiration. The high pitch and tubular quality are the important characteristics of bronchial respiration over the lung; the intensity may be, and frequently is, less than that of the ordinary vesicular murmur. This sign indicates solidification of a considerable or large portion of lung, and is found in croupous pneumonia, certain stages of phthisis, in compression of lung from large pleuritic effusions, large collections of air in the pleural sac, or the pressure of a tumor. As croupous pneumonia most frequently affects the lower lobes, bronchial respiration from this cause is commonly heard in the middle and lower portions of the back and sides. The intensity of the sound is usually proportional to the extent of solidification, and, as in croupous pneumonia the whole or greater part of the lobe is usually affected, the sign is most pronounced in this disease. In large pleural

effusions and some central consolidations the sound seems to come from a distance, the compressed lung being overlaid by fluid in the one case, and in the other a not too thick layer of healthy lung covering it. The inspiratory or expiratory murmur may sometimes be wanting, or the inspiration may be vesicular and the expiration bronchial in a more or less centrally localized consolidation, because the greater intensity of the expiratory murmur transmits the sounds farther.

Broncho-vesicular respiration partakes of the qualities of both the bronchial and vesicular type, one or the other generally predominating. It is called by many harsh or rude respiration. The pitch is raised and the expiration prolonged in proportion as the bronchial element is marked. The most distinctive feature is the combination of the vesicular and the tubular quality in the inspiration. The sign denotes partial solidification of lung, a degree not sufficient to produce bronchial respiration. The bronchial element is more or less marked according to the degree and extent of the solidification, its nearness or otherwise to the chest wall, and the thickness of the latter. It is heard in the resolving stage of croupous pneumonia where the resolution has made some progress, and from day to day the bronchial element lessens while the vesicular sound increases. The pitch gradually becomes lower and the expiration shortens. It forms a valuable sign in phthisis, giving evidence not only of the fact of consolidation, but of its degree and extent. It is also heard in interstitial pneumonia, hemorrhagic infarct, condensation of lung from pressure effects of fluid, air, or tumor. Sometimes the sign is only made out after considering the other physical signs. It must be remembered that normally the respiration over the right apex is less vesicular and higher in pitch than over the left ; in other words, it has more or less of the characters of the broncho-vesicular respiration.

Amphoric respiration denotes a respiratory sound with a metallic or musical tone or echo. It owes its name to the analogy it presents to the sound produced by blowing into a pitcher, decanter, or bottle. Whenever the respiratory sound has this intonation it indicates a space containing air which is not expelled with the expiration. Amphoric respiration may be heard with either inspiration or expiration, or both. When it accompanies one act alone it is most frequently the expiration, as it is here the loudest and most distinct. It is heard only in pneumo-thorax and in large pulmonary cavities. In pneumo-thorax to have the sign produced the perforation of the lung must be above the level of the liquid if any be present, and be unobstructed. Pneumo-thorax may exist, therefore, without this sign. To be produced in a pulmonary cavity the latter must have rigid walls and be as large as the fist, have a free communication with one of the larger bronchial tubes, and be situated close to the surface of the lung. Such cavities are usually found in the upper part of the lung in front, and the area of the sign is limited by the size of the cavity. The sign in pneumo-thorax is usually heard in the middle and lower regions of the chest.

Cavernous respiration is a sign described by Flint and accepted by the English and French, but not at present by the Germans generally. It is rarely met with. The characteristics which distinguish it are the entrance of air into a cavity with inspiration and its exit from the cavity with expiration. This passage of air into and from the cavity can only

take place where the cavity expands more or less in inspiration and collapses during expiration. Such cavities may exist in phthisis, or rarely in circumscribed abscess and gangrene of the lung. The inspiratory sound is neither vesicular nor tubular in quality; it is simply blowing. The pitch is low as compared with the bronchial respiration. The expiratory sound is of the same quality as the inspiratory, and it is lower in pitch. The duration is variable, as is also the intensity. The distinctive characters, then, are the quality and pitch, which are blowing and low respectively. It is oftener found at the upper part of the chest than elsewhere, occurring for the most part in phthisis, and its area is limited, corresponding to the size of the cavity. It is not constantly found in cavities with flaccid walls, and may be temporarily absent from the presence of liquid in the cavity or from obstruction due to secretion. Moreover, the cavity must be situated near the surface, and solidified lung must not intervene between it and the chest wall, as bronchial respiration will conceal it. In some cases of perforation of lung with pneumo-thorax air may pass to and fro through the perforation and give rise to cavernous respiration, but, as a rule, amphoric respiration is heard here.

Broncho-cavernous respiration is very rarely heard. There may be a mixture of the cavernous and bronchial inspiration and expiration, or a bronchial inspiration and cavernous expiration, or an inspiration the first part of which is bronchial and the last part cavernous, as has recently been described by the Germans under the title "metamorphosing respiration."

A vesiculo-cavernous respiration is heard where a cavity is surrounded by healthy lung, which is also rare.

Rhythm.—The rhythm of the respiration may be modified as follows: the inspiration may be shortened, the expiration may be prolonged, or the respiration may be interrupted, jerking, wavy, or cogwheel.

Shortened inspiration may occur from two opposite conditions—either from partial filling up or dilatation of the pulmonary alveoli. In the former case it is incident to croupous pneumonia; the pitch is high and the sound tubular. The inspiratory sound ends before the inspiratory act is completed. In the other case the sound does not begin with the inspiratory act; it is deferred. The quality is more or less vesicular, and the pitch is not notably raised. This form is heard in vesicular emphysema, and is a valuable diagnostic mark.

Prolonged Expiration.—In health the expiration varies in length in different persons. Sometimes it may be nearly as long as inspiration. The pitch and quality here do not differ from the normal sounds; they are low and vesicular respectively. Frequently, however, the pitch is somewhat raised and the quality a little tubular in the right apex, but if there be no other signs of disease, it must be considered normal for the individual. Prolonged expiration, with elevation of pitch, tubular quality, and other signs of disease, is always abnormal. It is bronchial respiration. It indicates solidification of lung. Prolonged expiration of low pitch and non-tubular quality usually is incident to emphysema, though it may be sometimes heard in bronchitis.

Interrupted respiration, also called jerking, wavy, or cogwheel respi-

ration, may occur in the inspiration or the expiration, or both. It is more common to find the interruption in inspiration, which is broken up into two, three, or more portions. Without accompanying alterations of pitch and quality the sign has little diagnostic worth. It is sometimes found in healthy persons at the top of the chest, oftener on the left side. When of pathological origin the pulmonary alveoli of the apex may be partially infiltrated, and the mucous membrane of the finer bronchi swollen, so that the air enters less readily, and the lungs are later in expanding, than in the healthy portions adjoining, so that thus the respiration becomes jerking or interrupted. Repeated long breaths or cough may cause it to disappear for a while, but it returns in a short time. Interrupted inspiration indicates merely the existence of some obstacle to the entrance of air into the pulmonary alveoli: this obstacle may be of short duration, and is then of no account; in other cases it persists for a considerable time, and is then to be accepted as indicative of incipient catarrh of the apex of the lung.

New or Adventitious Sounds.

Râles.—When the respiratory organs are normal the only sounds heard in respiration are the ordinary breath sounds, as the mucous membrane lining the air passages is smooth everywhere, and no more secretion is formed than is necessary to keep the parts moist. When, however, the membrane becomes uneven and rough anywhere from swelling, and more secretion takes place, the accessory sounds called râles are added to the respiratory murmur. Râles are classified according to the localities in which they occur as laryngeal, tracheal, bronchial, cavernous, vesicular, pleural, indeterminate. They are further divided into coarse and fine, dry and moist.

Laryngeal or *tracheal râles* are either moist or dry. Moist râles are heard in these localities when mucus or other liquid has accumulated there. Bubbling sounds are produced, which are loud and usually heard without the stethoscope. The most typical râles of this variety are heard in the so-called death rattle incident to the moribund state. They are also heard when fluid has accumulated during a condition of insensibility to the presence of fluid, as in ether narcosis or coma from any cause, or they denote an inability to remove the cause by expectoration. Dry râles are caused by spasm of the glottis or stenosis at or below the glottis, from œdema, exudation, the presence of a foreign body, the pressure of a tumor, paralysis of laryngeal muscles, strictures following ulceration, and the like. The sounds are dry and whooping, crowing, snoring, whistling, wheezing, and so forth. They are also heard without the aid of the stethoscope. The sound produced by spasm of the glottis is heard in laryngismus stridulus, pertussis, croup, and in aneurysm or other tumor irritating the recurrent laryngeal nerve. Paralysis of the laryngeal muscles gives rise to a dry sound, as does also pressure of an aneurysmal or other tumor or cicatricial contraction involving stenosis of the trachea; the respiration is here termed stridulous.

Bronchial râles may be either moist or dry. Moist bronchial râles are bubbling sounds produced in bronchi of various size, and are hence

described as coarse, medium, and fine according to the calibre of the tubes. They may be imitated by blowing into a syrup or glycerin through tubes of different size. Coarse bubbling râles sometimes occur in acute bronchitis of the larger tubes, more frequently in the chronic form, however, and in children who do not expectorate. They are more common in bronchorrhœa and in profuse hemorrhage. The râles are heard on both sides of the chest with either inspiration or expiration, or both. Medium bronchial râles are more frequent, and are heard in the conditions mentioned above.

Fine bubbling sounds, called also subcrepitant râles, are produced in the smallest bronchial tubes. They are incident to bronchitis of the smaller tubes, and are heard in bilateral, pulmonary œdema, the resolving stage of pneumonia, heard only over the affected area, hemorrhage, and the different stages of phthisis. They are heard with both acts of respiration, though most frequently during inspiration. The moist bronchial râles of whatever variety vary in pitch according to whether the surrounding lung is solidified or not; in the one case they are high, in the other low, and, as they often obscure the breath sounds, it is well to accustom one's self to recognize differences of pitch in order to estimate the condition of the lung where the râles occur.

Dry bronchial râles produce a sound which is either snoring, wheezing, whirring, humming, clicking and interrupted, or hissing or whistling. The former are called sonorous râles, and are low pitched; the latter sibilant râles, and are high pitched. As a rule, sonorous râles are produced in large bronchial tubes, and the sibilant in small ones. Both may occur at the same situation, the sibilant râles being heard during inspiration and the sonorous ones during expiration; but the râles may change from one form to the other several times within a short interval in the same locality. It is well to bear in mind that the sonorous râle is often mistaken for a pleural friction sound by inexperienced practitioners. The diagnostic inference from the presence of dry râles is that the mucous membrane of the bronchi is swollen in the locality where they are heard, and covered by a scanty and viscid secretion. The dry râle is heard best in asthma and some cases of emphysema. Dry râles are loud and intense, and may frequently be heard at a little distance from the patient. Single râles, moist or dry, are at times heard in healthy persons on sudden or prolonged inspiration and expiration in different parts of the chest. They have no special significance in such cases without accompanying physical signs.

The *vesicular* or *crepitant râle* is the only one which arises in the air vesicles. It is a very fine, dry sound, and may be imitated by twisting a small lock of hair between the thumb and finger near the ear, or by covering the palms of the hands with soap lather and, after pressing them firmly together, separating them near the ear, or by throwing a little dry table salt on a hot stove or burning coals, or by placing the end of the stethoscope over a part of the chest covered by hair and listening to the respiration. In the latter case the sound may be eliminated by wetting the hair with water, when it will adhere closely to the surface of the chest. The crepitant râle is heard only at the end of an inspiration, usually a forced one; it is a dry crackling and not a bubbling; it is made up of sounds all of which are even, and may be best desig-

nated as a shower of crackles heard at the end of a forced inspiration. The crepitant râle is specially characteristic of croupous pneumonia in the early or late stages, when the viscid walls of the alveoli are separated during inspiration; it is rarely heard during the stage of consolidation. By the time a case of croupous pneumonia comes under medical observation the crepitant râle has usually disappeared, the alveoli being more or less filled with exudation. When resolution has begun and progressed a certain distance the crepitant râles may reappear together with the subcrepitant râles, the latter being characterized by their unevenness and by their being heard also during expiration as distinct bubbling sounds. The crepitant râle also occurs in some cases of phthisis, especially at the apex. It is generally conceded that the sound is due to the sudden separation, through the inspiratory act, of the walls of the alveoli previously in apposition.

The *cavernous* or *gurgling râle* occurs in a pulmonary cavity of considerable size containing liquid and communicating freely with a bronchus. The sound is a large bubbling like the boiling of water in a flask, and is accurately expressed by the term "gurgling." It is sometimes high pitched and amphoric with a musical or metallic quality, but it is usually low in pitch. It is usually somewhat intense, and almost invariably heard at or near the summit of the chest during inspiration, though it may be propagated to a distance. It is of importance in the diagnosis of the advanced stage of phthisis.

Pleural râles are heard when the surfaces are roughened, giving rise to the pleural friction or rub. This has an intensity which varies greatly from the lightest rubbing sound, just audible to the skilled listener, to one of a shuffling, grating, or creaking character, which may be heard at a distance and be readily felt by the hand applied to the chest. The friction sound may be heard with both inspiration and expiration, or with either alone. It is near the ear, and can often be intensified by pressure with the stethoscope. The loud, intense sound is frequently broken or interrupted during the act of inspiration or expiration, while that of low intensity is ordinarily continuous. The pleuritic friction sound usually denotes pleurisy. The sound is rarely heard in the beginning of pleurisy, because the patient restrains his breathing on account of the pain produced and also because the surfaces are not yet sufficiently roughened to produce it. When effusion has taken place there is no sound, because the surfaces are separated by the fluid. It is usually first appreciated when absorption begins, and the more rapidly this progresses the greater the area over which the sound is audible. The sound may be heard for a short or long time, according to whether the surfaces become agglutinated early or only after the lapse of considerable time. If there be no fibrinous exudation on the pleura, friction is wanting, so that it is never heard in cases of transudation. In dry pleurisy or pleurisy without effusion it is frequently heard in circumscribed areas which are often painful.

There is always coexisting pleurisy where a pneumonic consolidation approaches the surface of the lung, and often the friction sound may be heard in these cases.

Circumscribed friction sometimes depends on the inflammation set up by broken ribs, or disease of ribs or cartilages, or new growths.

Slight friction at the apex of the lung is one of the accessory signs of phthisis, and here denotes a circumscribed dry pleurisy, which has diagnostic significance.

Inequalities of the visceral surface have sometimes been found to produce the sound, as miliary tubercles or cancerous nodules.

Metallic tinkling may be produced by the respiratory acts and by the voice or cough under conditions about to be described. It consists of a series of tinkling sounds, high pitched, metallic, or silvery in tone, which may occur during either inspiration or expiration. It may be a single sound or several, and is usually heard at irregular intervals and not with every act of respiration. It is found only in cavities of a certain size, at least as large as the closed fist, which are surrounded by walls of uniform density and are situated near the surface of the lung. It is also heard in some cases of pneumo-hydrothorax with perforation of the lung. There must be air and liquid in the cavity capable of being set in vibration. Such metallic tinkling may be heard in the stomach, but by exercising care it will not lead to mistakes.

Splashing or *succussion sounds* may be obtained over the healthy stomach more or less distended with air and liquid when the patient is shaken or shakes himself. The sound may be heard at a distance or when the stethoscope is placed over the abdomen. The same thing occurs over the chest in certain pathological conditions—namely, in pneumo-hydrothorax and pyopneumo-thorax. The sound is often high pitched and amphoric. It is like the sound produced by shaking a bottle partly filled with liquid.

Indeterminate Râles.—Crumpling and crackling sounds are sometimes heard over the chest which cannot be referred to any special physical condition. If they are limited to the upper part of the chest, and especially to one side, very soon other signs may occur which point to incipient phthisis.

THE VOICE SOUNDS IN DISEASE, both loud and whispered, may be diminished or increased in intensity. They are diminished or entirely suppressed by all conditions which impair or stop the transmission of vibration from the larynx to the thorax. The most typical examples of this diminution or even suppression of voice sounds is met with in pleuritic effusion, empyema, hydro-thorax and pneumo-thorax, where the lung does not come in contact with the walls of the chest. In effusions which partially fill the chest with liquid there is diminution or suppression from the level of the fluid down, while above the level of the liquid the resonance is increased from condensation of the lung. The level varies with changes in the position of the body where pleural adhesions do not confine the fluid.

Diminution or suppression may follow complete solidification of the lung and bronchial tubes in croupous pneumonia; it is also found in pulmonary œdema and over an intra-thoracic tumor. It is, moreover, met with over a limited area in abscess of the lung before evacuation of the pus. Obstruction of a bronchial tube diminishes or suppresses the resonance in the entire area to which it is distributed, according to the degree of obstruction.

Bronchophony.—Increase in the vocal resonance indicates a condition favorable to the transmission of sound waves, such as is found in com-

plete or considerable consolidation of lung. The increase beyond a slight degree is called bronchophony, and the characteristics of this sign are, compared with normal vocal resonance, concentration, nearness to the ear, and more or less elevation of the pitch—conditions which suggest the laryngeal voice sounds, except that the strong thrill or concussion is absent here. Bronchophony is heard in croupous pneumonia, phthisis, fibroid phthisis, condensed lung in pleuritic effusion, from the same thing due to air in the pleura, pressure of a tumor, or coagulated blood in the alveoli. It is a sign which accompanies bronchial respiration, but the degree of solidification necessary to produce bronchophony is less than that needed for the production of tubular respiration, so that bronchophony may accompany a broncho-vesicular respiration as well. This is well illustrated in the terminal or resolving stage of croupous pneumonia, where the bronchophony far outlasts the bronchial respiration.

Whispering bronchophony corresponds in character to the expiratory sound in bronchial respiration, and has the same significance.

All exudations are not alike in their resistance to the transmission of voice sounds. Bacelli has claimed that the vocal vibrations easily penetrate the more fluid and homogeneous effusions, and that a whisper even may be heard under favorable conditions, while the sound does not pass at all or only with difficulty through fibrinous, purulent, or bloody exudations, because the fibrin or corpuscles scatter the waves and they are not heard. The whisper, he claimed, could be heard most distinct at the base of the exudation while the face of the patient was turned to the other side. This is whispering bronchophony, and it is heard in the following conditions as well: over cavities and where the alveoli are filled with fibrinous or caseous material. On the other hand, the claim is made, and apparently substantiated, that the Bacelli phenomenon does not occur in every serous exudation (pleurisy), and that it is found in rare cases in purulent and sanguineous exudations.

Ægophony is a modification of bronchophony, the pitch and concentration being like the latter, while it is distinguished from it by its apparent distance from the ear and its tremulous or bleating tone, resembling that of a goat, the nasal sound of the voice when the nostrils are closed, or the sound when a comb is covered with tissue paper and held close to the mouth while speaking. It is commonly met with in cases of moderate pleuritic effusion where the lung, covered by a thin layer of fluid, is condensed underneath sufficiently to give rise to bronchophony. The sign sometimes occurs also in cases of pneumonia with pleuritic effusion. Ægophony is also sometimes heard in healthy women and children, in the latter especially between the shoulder blades. And finally, when the stethoscope is placed very lightly on the chest in auscultation, or only a portion of it touches the chest, or when the person examined speaks with the teeth close together, the same sound may be heard. It is not a sign of very great importance.

Increased bronchial whisper has the same significance as increased vocal resonance and broncho-vesicular respiration; it represents the same physical condition—namely, solidification of lung to a degree less than that which gives rise to bronchophony and bronchial respiration. The characteristics of the sign are—increase in intensity, a more or less

tubular quality, and an elevated pitch corresponding to the degree of solidification. The normal disparity between the two sides must be borne in mind, a greater intensity on the left indicating disease. The diagnostic application of the sign is in phthisis in the early stages.

Pectoriloquy signifies the transmission of the words to the ear, and may be either whispering or loud. It is an unimportant subdivision of bronchophony.

Amphoric voice, or *echo*, is identical with amphoric respiration in character, and is usually associated with it. It may accompany the loud voice or whisper, and is generally more marked with the latter. Its signification is the same as amphoric respiration—namely, a large pleural or pulmonary cavity with rigid walls in direct communication with a bronchus. The sound sometimes follows the voice; hence the term "echo."

Cough.—The act of coughing may be used as an aid in auscultation in several ways:

1. The inspiration immediately after repeated coughing is deeper and the respiratory murmur is more intense.

2. Cough, especially if followed by expectoration, may remove mucus which previously obscured or obstructed bronchial tubes, so that the true character of the respiration may be obtained.

3. Râles are brought out or intensified by coughing. Any fluid in cavities or bronchi is set in more violent vibration by this means than by ordinary respiration; its location is often changed thereby to more confined spaces and the râles are intensified and increased in number.

4. If there are conditions in the lung which give rise to bronchial respiration, bronchophony, and whispering bronchophony, the cough is of a bronchial character. Over a cavity of some size near the surface the cough gives rise to a forcible shock, which is highly significant. The cough may have an amphoric character, corresponding to the amphoric voice in appropriate conditions. Cavernous gurgling and metallic tinkling may also be more distinctly heard with the cough than with the respiration.

Sputa.—The secretions of the respiratory organs which are expelled by the acts of coughing or hawking are called expectoration or sputum. They are secretions of the pharynx and of the larynx, which are usually got rid of by hawking, and those of the trachea and lungs, which are expectorated by the aid of coughing, in case they are not swallowed, as is usually the case in children, or they are raised to the upper parts of the respiratory apparatus by the cough and are later expelled by hawking. Very commonly substances from the mouth and naso-pharyngeal space or stomach are mixed with the sputum, but these are not included in this description.

The sputum should be collected in spit-cups of crockery or glass for the purpose of examination. This examination is macroscopic, microscopic, bacteriological, and perhaps chemical.

I. Macroscopic Examination.—The sputum is considered in reference to its amount, appearance, and consistency, color, smell, and taste. Its reaction is always alkaline when not mixed with gastric contents. After examination in the spit-cup it is best poured into a plate with a dark background for further investigation.

1. *Amount.*—The amount is scanty in affections of the throat and larynx, in the beginning of acute bronchitis, and in acute croupous pneumonia. It is more abundant in the later stages of acute bronchitis, in chronic bronchitis, and in phthisis. It is very abundant, a litre or more in a day, in bronchorrhœa, in phthisis with cavities, and bronchiectasis. In the latter disease it is often expectorated in large quantities in paroxysms of coughing. Large quantities may be expectorated at once in perforation of abscesses, perforation of serous and purulent pleural exudations, and of ecchinococcus cysts in the lungs.

2. *Appearance and Consistency.*—A mucous, purulent, muco-purulent, sanguineous, and serous sputum is recognized according to the predominance of the one or the other of these elements.

Mucous sputum is tenacious and stringy from its richness in mucin. Its color varies and its quantity is small. It is secreted in the beginning of acute catarrhs of the mucous membrane of any part of the respiratory tract.

Purulent sputum is generally fluid and flows together in the spit-cup from its poverty in mucin. It is derived from cavities, bronchorrhœa, the perforation of an abscess, or empyema into the bronchi.

Serous sputum is very liquid and foamy. It occurs in pulmonary œdema and in perforation of serous pleural exudation into the lungs.

Muco-purulent sputum is a mixture of the two kinds, which gives the name. The pus is either uniformly mixed with mucus, or single clumps of pus of various size, from small flocculi to great balls, are bedded in mucus. According to the consistency of the mucus one distinguishes it as globular, nummular, or confluent sputum. This kind of sputum is the most common. It occurs in chronic bronchitis and all diseases of the lungs in which chronic bronchitis occurs, especially phthisis, and in the later stages of acute bronchitis.

Bloody sputum consists largely or wholly of blood. The quantity may be very considerable, so that in the course of a short time 500 or 1000 cubic centimetres of blood may be coughed up. The blood is usually bright red of the so-called arterial character, and is often intimately mixed with foam, being fluid or more or less coagulated. The more rapid and abundant a pulmonary hemorrhage is, the more likely is it to have come from a large artery.

Under some circumstances it is very difficult to distinguish a pulmonary from a gastric hemorrhage. The following points are to be considered: In gastric hemorrhage the blood is dark, venous, in clumpy clots without air bubbles, and has an acid reaction usually, and is, moreover, mixed with gastric contents, frequently bits of food, while in pulmonary hemorrhage it has an arterial look and the masses of blood contain air; the reaction is alkaline; microscopic examination shows elements derived from the lung (ciliated epithelium and elastic fibres perhaps). A pulmonary hemorrhage may have been so rapid and abundant that some of it was swallowed and afterward vomited, or blood may have come from the nose, pharynx, or mouth. A careful consideration of the history will usually readily differentiate these, however.

Pure bloody sputum may appear in all destructive processes of the lungs—most frequently in phthisis, gangrene of the lung, and sometimes

abscess. It may also follow ruptured aneurysms, echinococcus cysts, and certain cardiac diseases and embolism. Excessive paroxysms of coughing may give rise to bronchial hemorrhage or putrid bronchitis in rare cases.

Small quantities of blood may appear as red lines or points derived from the nose, throat, mouth, larynx, and tingeing the sputum or intimately mixed and in greater amount, as in the rusty sputum of croupous pneumonia. Larger clumps may be found in the muco-purulent expectoration of tuberculosis.

3. *Color.*—The color is clear as glass in pure mucous and serous sputum. It is gray in mucous and muco-purulent sputum from admixture with dust and particles of coal. Muco-purulent sputum is yellow and pure purulent sputum is greenish yellow. When mixed with blood the sputum is red, as described above.

4. *Odor.*—Sputum may be odorless or have a mawkish smell. It has a bad odor only when it stagnates, as is seen toward the end in cases of phthisis. Pure purulent sputum, as in abscess of the lung and in perforation of a purulent pleuritic exudation into the bronchi, frequently has a sour smell suggestive of buttermilk. It takes on a stinking character in putrid bronchitis and gangrene of the lung. On standing freely exposed when slight this odor may largely disappear, but, on shaking or emptying it, it is again perceived. The odor, on the other hand, may be so powerful as to scent the entire room or house.

5. *Taste.*—The taste is of slight diagnostic importance, and is usually described by the patient as salty or sweetish. Bloody sputum usually tastes salty.

II. Chemical examination has shown the presence of mucin, albumin, and globulin, fats, sugar, blood, biliary coloring matter, pigment, and ferments in the sputum. The qualitative examination for these substances is superfluous, for they are either constantly present in expectoration, or, in case they occur only in certain diseases, their presence can be determined more easily in some other way. Quantitative examination has thus far not given results of value.

III. Microscopic Examination.—1. *Red blood corpuscles* are found in large numbers in case there is blood in the sputum, as in croupous pneumonia, tuberculous hæmoptysis, infarct of the lung. They are usually well preserved, but often have given up their coloring matter and then appear as pale rings.

2. *White corpuscles* are found in all sputum. Their number depends on the quantity of pus in the expectoration. They are scanty in mucus and very abundant in pure purulent sputum. They are either well preserved or have become fatty; they often contain pigment granules.

3. *Epithelium* of three varieties is found in the sputum: (*a*) squamous epithelium from the mouth; (*b*) ciliated epithelium from the mucous membrane of the trachea, bronchi, and nose: such cells are rather few in number, and often their ciliæ have become detached; (*c*) alveolar epithelia, which are elliptical or polygonal cells measuring 20 to 50 micromillimetres, contain one or several nuclei, are finely granular, and often contain a fat drop or two, or even are wholly fatty. Sometimes they contain drops of myelin or pigment granules, which pigment is black from coal-dust or brown from blood coloring matter or iron.

These alveolar cells are found in the most different affections of the lungs, and their source is still *sub judice*.

4. *Particles from tumors*, such as carcinoma, sarcoma, and so forth, are very rarely found in the sputum with their characteristic arrangement of cells.

5. *Organic Masses.*—Elastic fibres from the lung may be found. They are highly refractive fibrils, frequently with an alveolar arrangement, which resist the action of potassic hydrate. They are present in destruction of lung tissue, as in phthisis and abscess of the lung. They are almost always absent in gangrene of the lung, because they are destroyed by the products of putrefaction. Fragments of lung tissue are found in the sputum in gangrene of the lung, and may often be seen with the unaided eye on a dark background. Under the microscope they are recognized from their alveolar arrangement.

6. *Fibrinous casts* of the bronchi are sometimes found. They are twig-like branches, usually very small, and may occur in the sputum of croupous pneumonia or fibrinous bronchitis. So-called Curschmann's spirals may be found in bronchial asthma and other lung diseases. They are spiral bodies perhaps one millimetre thick and several centimetres long, having a homogeneous thread in the centre with a mucin mass wound around them, holding in their meshes numerous white corpuscles and frequently Charcot-Leyden octahedral crystals. (See Fig. 18, p. 171.)

7. *Mucus* is found in all sputum more or less abundantly, usually in threads which are rendered distinct by acetic acid. More or less fatty white corpuscles and epithelium or some free fat drops are also found, and fine granular detritus of degenerated cells, perhaps also free pigment.

8. *The crystals* which have been described are—1. Charcot-Leyden colorless octahedra, insoluble in ether and alcohol, soluble in acids, alkalies, and warm water. They are the phosphatic salt of the base C_2H_5N, and occur in bronchial asthma and rarely in other forms of bronchitis. 2. Hæmatoidin crystals of red color occurring as needles or rhombic prisms, either free or in white corpuscles. They are indicative of a previous hemorrhage from abscess of the lung or other cause. 3. Cholesterine crystals, rhombic, highly refractive plates, soluble in ether and alcohol, insoluble in water, acids, and alkalies. On treatment with dilute sulphuric acid they become yellow to red on their edges, and by addition of tincture of iodine they become colored violet and blue. They occur in phthisis and abscess of the lung. 4. Fat crystals, which are long, pointed needles readily soluble in ether, insoluble in water and acids. Found in gangrene of the lungs, putrid bronchitis, phthisis, abscess, bronchiectasis. 5. Triple phosphate coffin-lid crystals found in gangrene and abscess. 6. Calcic oxalate crystals are also found rarely.

9. *Animal Parasites.*—The only one which comes under consideration is the echinococcus. It is recognized under the microscope by its homogeneous striped layers and the hooklets.

10. *Vegetable Parasites.*—1. Tubercle bacilli of Koch. They are cover-glass preparations stained by the Ehrlich or Ziehl-Neelsen method described under the head of Tuberculosis. They occur in the expectoration only in tuberculosis, and are absolute proof of its presence. On

the other hand, their continued absence after many trials is only presumptive evidence against tuberculosis. Their amount and appearance have no prognostic value.

2. Diplococci pneumoniæ (Fränkel-Weichselbaum). These may be found by two or three hours' staining in the Ziehl-Neelsen fluid and prolonged washing in alcohol. They are very small cocci which lie singly or several in a row, and each surrounded by a capsule which is either very feebly or not at all stained by the coloring matter. They occur in large numbers in croupous pneumonia, but also a few in the normal secretions of the mouth, so that microscopic detection has limited diagnostic value. (See Pneumonia, p. 199.)

3. Sarcini pulmonum (Virchow). These are cocci which lie together in fours, and may be recognized readily in unstained preparations. They occur in the most varied extensive ulcerations of the lung. They are stained according to Weigert's method.

4. Leptotricheæ are large bacilli which occur chiefly in the form of threads. They are specially found in putrid bronchitis and gangrene, and are stained violet by the iodine-iodide-of-potassium solution, while they take up the aniline colors feebly.

5. Actinomyces bovis (Harz). These micro-organisms are globular-shaped masses of yellow color, which in the centre consist of numerous fibres or rays which toward the periphery end in a club-shaped or dog-eared processes. They are readily recognized in unstained preparations with low powers of the microscope. These balls often lie joined in large masses, and can be seen then with the unaided eye as small organisms like grains of sand. They are found in the sputum in actinomycosis if the abscess sits directly in the lung or their pus can empty into the bronchi or trachea.

Other organisms rarely occur, and have no diagnostic significance.

Bacteriological examination requires an expert in such investigations.

BRONCHITIS—ACUTE, CHRONIC, PLASTIC; BRONCHIECTASIS.

By A. LAWRENCE MASON, M. D.

ACUTE BRONCHITIS.

Definition.—An acute catarrhal inflammation of the bronchial mucous membrane, varying in degree and extent. It is usually bilateral, and may affect any or all of the tubes down to the ultimate bronchioles. These, however, in the so-called capillary bronchitis are not involved without impairment of the corresponding alveolar structure.

Etiology.—The causative agencies are very numerous, as acute bronchitis is one of the commonest of ailments, and an attack may usually be traced to some recent exciting factor, although the element of predisposition has much to do with the etiology. The relation of morbid germs to the production of this disease must also be considered.

The most important exciting causes are cold and sudden variations of temperature, especially when combined with high winds and dampness. These conditions are met with in most northern latitudes, more on the seaboard than in inland regions; therefore in our Eastern and Middle States at certain seasons of the year bronchitis is widely prevalent. This prevalence in our own country, as in Northern Europe, increases steadily from the minimum at midsummer to the maximum in midwinter, with occasional variations in mild seasons or in times of epidemic, such as the late visitations of influenza.

The seasonal relations of bronchitis appear to differ somewhat from those of pneumonia, inasmuch as the latter disease prevails most extensively a month or two later or in the early spring. While "catching cold" does not meet with so much favor as it formerly did as a satisfactory explanation of all cases of bronchial catarrh, still there are very many instances in which this cause and its effect are perfectly apparent, although due weight must be given to other predisposing and exciting influences. As to the manner in which cold acts in producing bronchitis, whether directly through the effect of the inspired air upon a sensitive mucous membrane, through a cold draught striking the person, or through the chilling of the surface incident to wet feet or clothing, it is sufficient to say that in any or all of these ways a susceptible individual, with or without lowered vitality, may contract bronchitis. There is no more frequent cause than going from a hot, ill-ventilated house into the cold, damp air; and to this mode of exposure we are especially subject in America, owing to the powerful furnaces and steam radiators by which our buildings are warmed, and for which there is a public demand, although such ill-regulated heating is far from wholesome. It

is not improbable that the prolonged inhalation of overheated, noxious air is in itself a cause of bronchitis.

Workers in dusty atmospheres, such as mill-hands and metal-workers, may have frequent bronchial attacks through the irritant effect of inhaling the minute particles. The same is true of the inhalation of pungent gases like chlorine and the fumes of ammonia or sulphuric acid, but frequent exposure to these influences sometimes creates tolerance.

The effect of the pollen of plants, emanations from hay, and the dust of the railroad will be considered in connection with Hay Fever, p. 178.

The exciting cause of bronchitis may also be of internal origin in connection with certain acute and chronic diseases, of which measles is the most conspicuous. Inflammation of the respiratory mucous membrane is one of the earliest symptoms of that affection. Bronchitis is a common accompaniment of typhoid fever, and less frequently of scarlet fever, smallpox, typhus, and other zymotic diseases, in which the catarrhal symptoms are the expression of blood contamination by morbific elements. Gout, rheumatism, diabetes, and syphilis may be classed in the same category. Coryza and bronchitis are provoked in certain persons by small doses of the iodide of potassium, owing to an idiosyncrasy which renders them intolerant of this drug. Under these circumstances the inflammation is often of a very acute type, whereas the secondary bronchitis of typhoid and of the other diseases mentioned above is more frequently of the subacute variety.

The poison of influenza, however, manifests itself in many cases by a severe, sometimes violent, bronchitis, while in others the respiratory apparatus is relatively unimpaired. The further etiology of acute bronchitis relates to its predisposing or more remote causes, and under this heading may be included the age, the general health, and the habits or mode of life of the individual.

The frequency of this disease in young children and in the aged is very noticeable. While in the former case it is often coincident with the process of teething, it is to be borne in mind that infants are very susceptible to all deteriorating influences and that their respiratory organs are especially vulnerable. Nasal catarrh, hypertrophied tonsils, and adenoid growths, with the habitual mouth-breathing thereby induced, are no doubt fruitful sources of repeated bronchial attacks, and the indoor lives which such delicate children lead render them unable to bear even moderate exposure.

At the other extreme of life, besides the age and lack of resistance, the various diseases and degenerative processes from which old people suffer are causes which make them peculiarly liable to dangerous attacks of acute bronchitis; and at all ages cardiac lesions, Bright's disease, tuberculosis, cancer, and other debilitating cachexiæ create a susceptibility to bronchitis so marked in some instances that extreme care is necessary to prevent its frequent occurrence.

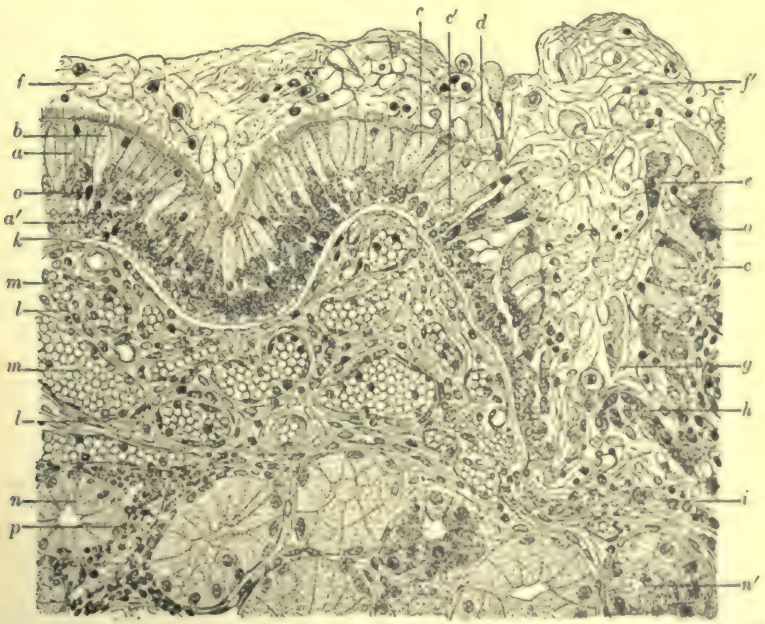
Self-indulgent habits and lack of outdoor exercise, by promoting digestive disturbances and the inability to stand much strain or exposure, predispose to bronchitis, a very common affection in regular drinkers which tends to assume a more chronic form in habitual drunkards. The irritant effect of tobacco is usually confined to the pharynx and larynx, as is the "ear cough" which sometimes attends aural lesions.

Finally, it appears that the atmospheric influences and the types of population in manufacturing towns and large cities are more conducive to the spread of bronchitis than are the conditions which prevail in rural districts, and that the tendency is to repeated attacks year after year during the colder months in persons who have acquired a susceptibility to this disease. It may occur in epidemics. An hereditary predisposition to attacks of acute bronchitis no doubt exists in some persons, but chiefly as a secondary manifestation in asthmatic, gouty, or rheumatic subjects.

While, then, the symptomatic character of bronchitis in many instances should cause us to regard it as a secondary condition rather than as a primary disease, this is not always so, and not infrequently the affection begins and runs its course as *bronchitis simplex* without constitutional cause and without complications.

PATHOLOGY.—In ordinary uncomplicated bronchitis the inflammation is limited to the trachea and the large or middle-sized tubes. This is termed tracheo-bronchitis. Catarrh limited to the large tubes has little effect upon the lung function. When the smaller tubes are

FIG. 14.



Histological lesions of acute bronchitis (after Ziegler): *a*, ciliated epithelium; *a'*, deep layer of stratified round cells; *b*, goblet cells; *c*, superficial cells which have undergone mucous degeneration; *c'*, cells in which the nucleus and protoplasm have undergone mucous degeneration; *d*, desquamated mucous degenerated cells; *e*, desquamated ciliated epithelial cell; *f*, mucus on surface; *f'*, fibrillated mucus containing pus corpuscles; *g*, excretory duct of mucous gland filled with cells and mucus; *h*, desquamated epithelium of excretory duct; *i*, epithelium of duct in position; *k*, hyaline basement membrane; *l*, connective tissue of mucosa, in part infiltrated with round cells; *m*, dilated bloodvessel; *n*, acinus of mucous gland filled with mucus; *n'*, acinus of mucous gland empty; *o*, wandering cells in epithelial interstices; *p*, round cell infiltration in periglandular connective tissue.

involved, however, it is of the greatest significance, not only because of the danger of direct extension to the parenchyma, but also because the

swelling of the mucous membrane and the accumulation of secretion narrow or close the bronchi and may produce atelectasis.

However the disease may be produced, and however severe its onset and course may be, there is no essential difference in the phenomena produced other than in the degree and the rapidity of their development. The gross manifestations are reddening and swelling of the mucous membrane, which is covered with mucus and muco-pus. The smaller bronchi are filled with the same material, which on section oozes from them.

Bronchitis begins with engorgement of the vessels in the inner connective tissue layer, causing redness and swelling. At this stage there is little or no secretion of mucus, and the mucous membrane subjectively feels dry and irritated. Exudation of serum and leucocytes from the vessels soon follows. These at first tend to collect beneath the basement membrane and about the glands. As the exudation continues, however, the basement membrane becomes highly œdematous and leucocytes pass through it toward the surface. At the same time, the superficial epithelial cells swell, and many ciliated cells are converted into goblet cells with profuse discharge of mucus. Up to this time there has been no proliferation or desquamation of the epithelium, but by the second day the epithelial cells proliferate and desquamate. The same process also takes place in the lining cells of the mucous glands, while the passage of leucocytes to the surface is more rapid and profuse. After a time, if the inflammation continues, the connective tissues become involved, the process finally extending even to the adventitia, where cell-proliferation and a very marked infiltration with leucocytes occur. The bronchial glands often become and remain enlarged, thereby causing a predisposition to recurrent attacks.

If the inflammation ceases, there is a gradual return to the normal state, the process closely resembling that of healing on the surface of the body. On the other hand, it may increase, becoming purulent and even gangrenous. When the process extends to the smaller bronchi, as the affection is general and bilateral, there is great obstruction to the ingress and egress of air. An amount of swelling which in the larger tubes causes but little impediment to respiration in the smaller ones may result in complete stenosis and consequent atelectasis. The secretion is also more difficult to dislodge and may entirely block the tubes.

When the ultimate bronchioles are invaded the alveolar structure does not escape, but broncho-pneumonia ensues.

Capillary Bronchitis.—The capillary tubes are no longer regarded as the seat of a catarrhal inflammation which can with propriety be termed "capillary bronchitis" as a pathological entity, since the process cannot extend to the terminal bronchioles without a corresponding lobular involvement. Morrill, in Keating's *Cyclopædia*, expressed this view. Similar opinions are held by Wilson,[1] who says that the process in question "is always a broncho-pneumonia," and by Osler, who regards it as "only a part, though a primary and important one, of broncho-pneumonia," stating that he has repeatedly found lobular hepatization in diphtheria within forty-eight hours of the onset of the acute bronchial symptoms.

[1] *An American Text-Book of the Theory and Practice of Medicine*, Pepper.

This is now the general view, and the term "capillary bronchitis," if used at all, must be understood to mean either a bronchitis of the finer tubes which does not reach the terminal bronchioles, as was set forth by Flint many years ago in his *Practice of Medicine*, or an acute suffocative broncho-pneumonia involving both the bronchioles and the alveolar structure. In either case the designation "capillary bronchitis" fails to identify the pathological condition, and had better be abandoned, as have been the vague terms "catarrhus senilis," "peripneumonia notha" (Sydenham), and "suffocative bronchial catarrh."

SPUTUM.—The sputum is composed of the secretion from the inflamed mucous membrane and varies much both in quantity and quality. In the beginning it is scanty and viscid, but as the disease progresses it becomes more abundant and fluid, and contains many pus cells and numerous large, round alveolar cells. When the finer bronchi are involved it often contains small mucous or muco-purulent casts of the bronchi. In very severe cases there may be a little blood, which, however, is of no especial importance.

BACTERIOLOGY OF ACUTE BRONCHITIS.—Osler says: "The affection is probably microbic, though we have as yet no definite evidence upon this point." Claisse[1] states that while in the secretion of the greater bronchi during catarrh a mixture of various forms of bacteria is usually found, the small bronchi ordinarily contain only a certain kind, sometimes Fränkel's pneumobacillus, very frequently streptococci. These exceptionally pass out of the bronchi into the blood and possess a varying virulence. He thinks that their virulence increases sometimes if they pass through several persons, and advances as proof the fact that in some epidemics the later cases are more severe. Experiments showed that neither the mechanical irritation of the mucous membrane of the trachea nor the introduction of bacteria alone caused disease, but that if the bacteria were introduced after trauma an intense catarrh developed in twenty-four hours. Hence the conclusion that in nature the process is similar, and that the lesion of the mucous membrane may result from—

1. Physico-chemical trauma;
2. Elimination of poisonous substances with the bronchial secretion—iodine, alcohol;
3. Toxic products of infectious diseases—typhoid fever, cerebro-spinal meningitis;
4. Exanthemata—measles, smallpox;
5. Direct extension from mouth and nose to air passages.

Rarely do the pathogenic bacteria wander out from the circulation into the bronchi.

Queyrat[2] considers that simple tracheo-bronchitis is a contagious disease, and not due to simple exposure. In favor of this view are epidemics and cases in the same family. In cover-slips from the sputum of tracheo-bronchitis, especially in the early days, he found great numbers of a special micro-organism. These appeared as large cocci, often in the form of diplococci, sometimes in short chains. They were most abundant in the early days, diminished by the end of the first week, and disappeared in two weeks. He cultivated eleven cases

[1] *Semaine médicale*, 1893, 38.

[2] *Gazette méd. de Paris*, 1893, II., iii.

on gelatin, and obtained either yellow or white colonies of cocci, whose number diminished with the progress of the disease. They were streptococci in bouillon cultures. He found these same micro-organisms in all the cases of tracheo-bronchitis that he studied. Hence he concludes: "The constant presence of these organisms, as shown by examination and by cultures, and the fact that they are very abundant at the beginning of the disease and diminish and almost entirely disappear in the last days, seem to indicate that they play a preponderating rôle in the etiology of simple tracheo-bronchitis, and that cold has only an accessory part."

Grün[1] claims to have found a certain peculiar bacillus in every case of bronchitis which he has examined. This he isolated on agar and gelatin. He calls it "the bacillus of acute bronchitis," and considers it to be the cause of bronchitis.

Christopher[2] says: "Bronchitis occurring in an acute infectious disease is but one of the symptoms of that disease, and is produced by the same poison which is producing the other symptoms. Whatever be the poison, its ultimate action must be a chemic one, so that it is possible to formulate the doctrine that in certain infectious diseases a chemic poison, acting, in all probability, through the medium of the central nervous system, is capable of producing the condition bronchitis." He thinks the bronchitis and broncho-pneumonia associated with gastro-enteritis are due to chemic absorption from the bowels, and that the bronchitis occurring in other diseases is undoubtedly secondary and due to local infection caused by retained or inspired secretions and foreign substances. The bacteria present are in the majority of cases streptococci, less often staphylococci.

Marfan's studies on the relation of bacteria to bronchitis are valuable. While regarding all bronchitis as infectious, this writer considers some forms as directly due to the specific microbes of the diseases to which they are secondary (measles, whooping cough), coming either through the air or through the blood, while in others the poison is hæmatogenous, as in gout, Bright's disease, diabetes, and typhoid fever. It is not thought that Eberth's bacillus directly attacks the bronchi. But in the absence of definite knowledge as to the organisms which cause many of the infectious diseases—smallpox, scarlet fever, and syphilis, for instance—it is at present impossible to say whether the germs of diseases or their toxic products are responsible for the bronchitis which attends them. In gout, rheumatism, and other depressing conditions the protective barriers of the system are weakened through derangement of vaso-motor innervation; congestion ensues, and microbic infection may in turn result.

Morse[3] thinks that, as the nose and throat almost always contain pathogenic cocci, it seems reasonable to suppose that they are continually present in the trachea and bronchi also, and probably are necessary for the production of bronchitis, cold and other agencies merely according an opportunity for their entrance. He regards it as improbable, however, that there is a specific organism for the disease in general,

[1] *Lancet*, 1891, i. 1424. [2] *Journ. of the Amer. Med. Assoc.*, 1893, xxi. 871.

[3] The writer is indebted to Dr. John Lovett Morse, of the Boston City Hospital, for researches in the pathogeny of bronchitis and asthma.

since the ordinary bacteria, especially streptococci, are the ones usually found. But the presence of Pfeiffer's bacillus in the bronchi appears to indicate that this specific germ causes the bronchitis of influenza, and the local presence of a specific micro-organism in pertussis is highly probable, although this has not yet been demonstrated. In measles, early typhoid, and variola it seems doubtful whether the bronchitis is caused by specific germs, by an eruption in the bronchi which affords an entrance to the ordinary bacteria, or by the elimination of toxic products, which causes local irritation and favors the invasion of bacteria.

Experiments bearing upon these points are wanting, although catarrhal symptoms have been observed to follow the injection of the blood of a patient suffering from measles into a healthy person.[1]

Symptoms.—Acute bronchitis may involve the trachea and larger bronchi alone or it may also extend to those of smaller calibre. In most cases its seat is in the windpipe and the principal branches on both sides. The inflammation often begins in the upper air passages with a cold in the head or a sore throat, and takes a descending course. Sometimes, however, the bronchial passages are directly attacked.

In bronchitis of ordinary intensity chilly sensations are common, with a moderate degree of fever, sometimes none at all. General malaise is often present, and there is more or less anorexia, thirst, and pain in the limbs according to the severity of the attack. A furred tongue and quickened pulse may be noticeable from the onset. Flushed face, headache, and fulness in the ears are frequent accompaniments. The bowels may be sluggish.

The local indications of the first stage are, besides coryza and pharyngitis, a feeling of dryness and constriction in the upper sternal region, with hoarseness and a hard rasping cough due to the inflamed laryngeal and tracheal membrane. Wheezing and dyspnœa may be present, but there is usually little impairment of the respiratory function in uncomplicated acute bronchitis that is confined to the larger tubes. The cough is sometimes paroxysmal in character, especially at night, causing insomnia.

In a few days, when exudation from the mucous membrane begins, the cough becomes looser and is attended by expectoration, which is in some cases thin, frothy, or blood-streaked, in others muco-purulent. In mild attacks the catarrh runs its course in a week or two, the sputa being more easily raised as the inflammation subsides and the cough gradually abates. But in many instances, from want of care, the patient "takes more cold," as it is said; there is a recurrence of the symptoms, and the disease may be greatly prolonged by repeated exacerbations, the process reaching farther and farther into the bronchial ramifications. Such attacks, returning in more prolonged form from year to year, eventually cause chronic bronchitis and its complications, which will be considered later.

Bronchitis of the medium-sized and smaller bronchi, which is not uncommon in children and in old persons, as well as in certain acute infectious disorders, especially measles, is attended by the symptoms enumerated above, often in a greater degree. The fever and pulse may

[1] Macintyre, *Lancet*, 1893, ii. 479.

be high from the beginning, although an asthenic type of disease is common in the aged, with a temperature near or below the normal. Dyspnœa is urgent and the expectoration insufficient for relief. The face becomes dusky. There may be great restlessness, inability to sleep, complete anorexia, and prostration. The cough is constant and harassing, straining the intercostals, the diaphragm, and the abdominal muscles to a painful and exhausting extent.

In old persons incontinence of urine with the acts of coughing aggravates the discomfort. At both extremes of life, through inability to clear the bronchial passages, the tubes may become plugged by mucus, and collapse of portions of the lung (atelectasis) may take place. In the worst cases there is complete exhaustion, delirium, or coma. The urine is concentrated, with excess of urates, and is often albuminous.

In the so-called capillary bronchitis, when the terminal bronchioles and pulmonary lobules are also involved in the inflammatory process, the above symptoms are further intensified to an alarming degree and often with great rapidity. The alæ nasi distend and dyspnœa is most urgent, even to the extent of orthopnœa, while the respiration may reach, in the very young, eighty or a hundred a minute. The countenance is cyanotic and anxious, the heart rapid and feeble, with engorgement of its right side, and the strength does not suffice to clear the air passages of the sticky mucus which fills their deeper portions. Such cases are very fatal through the acute suffocative broncho-pneumonia which develops. In children convulsions may supervene.

Physical Signs.—The physical signs in acute bronchitis vary with its extent. Percussion, vocal resonance, and fremitus are not materially changed. The respiratory murmur remains normal in quality, but may fail to reach portions of the lung when the bronchi which lead to them are plugged with mucus.

In bronchitis of the larger and medium-sized tubes at an early stage dry, sonorous, and sibilant râles may be heard bilaterally, varying in site, and sometimes disappearing after forced respiration or cough. Often loud wheezing sounds are produced over both front and back—to a marked degree when asthma coexists. At a later period moist bronchial or bubbling râles are heard when the tubes contain a serous or muco-purulent exudate, and they are coarser or finer in accordance with the size of the tubes in which they are produced. But the râles of bronchitis are very inconstant, and auscultation in many cases gives negative evidence only.

Complications.—Laryngitis is very frequently associated with acute bronchitis, and in some children there is a strong tendency to attacks of spasmodic (false) croup. The severer complications relate chiefly to the obstruction of the smaller tubes by plugs of mucus, and the consequent collapse of the corresponding portions of the lung (atelectasis), and to the occurrence of broncho-pneumonia when the catarrhal process extends to the bronchioles and pulmonary vesicles. These conditions rarely supervene except in young children and in old or feeble persons, and have been previously considered with regard to the characteristic aggravation of symptoms and the danger which they cause.

Bronchitis occurring as a complication of various predisposing affec-

tions may in turn excite a greater degree of activity or induce a further development of the primary disorder. It lowers the vitality, prevents sleep, saps the strength, and may become an important element in the prognosis of the chronic diseases or cachexiæ of which it is an intercurrent manifestation. In chronic cardiac lesions, for instance, especially those of the mitral valve, coincident bronchitis tends to impair the compensation, to promote dilatation, and to endanger life.

Sequelæ.—There are few sequels of acute bronchitis beyond the predisposition to repeated attacks which may be created. In some cases of whooping cough, and occasionally after severe bronchitis from other causes, there may be a general vesicular emphysema; and in very rare instances violent coughing may cause rupture of air vesicles and interlobular emphysema. Such an occurrence usually escapes notice and the air is soon absorbed. It may, however, pass through the mediastinum into the subcutaneous tissues of the body. In this manner the supra-clavicular spaces, the face, trunk, and extremities may become much distended, causing the patient to present a very dropsical appearance. But the characteristic crackling under the skin reveals the nature of the trouble.

Bronchitis is common in tuberculous subjects, but if phthisis follows acute bronchitis, as it appears occasionally to do, it is probable that through individual susceptibility the catarrhal state of the mucous membrane affords a suitable nidus for the specific bacilli of tuberculosis.

Diagnosis.—The diagnosis of acute bronchitis is based upon the history, symptoms, and course of the attack, sometimes with a predisposing ailment, and the presence of the physical signs that occur in this affection, with the absence of those which accompany pneumonia, phthisis, and other pulmonary diseases. As was before stated, the attack is usually the result of some exposure of which the patient was aware, or it is an incident in the course of some acute or chronic disease, to be determined by further inquiry. The diagnosis "acute bronchitis," when it is a secondary phenomenon, is unsatisfactory and imperfect, both as regards the prognosis and the treatment, unless we can arrive at a correct conclusion as to its cause. This is not always easy. In measles, for instance. the cause of the preliminary coryza and cough may not be clear for several days or until the eruption appears. Whooping cough is often regarded as simple bronchitis until the characteristic spasm occurs. The probability that one or the other of these diseases may be developing is increased by a knowledge of their prevalence in the neighborhood, but this seldom suffices for an absolute diagnosis in the first case in a family.

In gouty and rheumatic subjects, although they are especially prone to bronchial catarrh from slight exposure or none at all, the chief etiological factor may escape notice in the earlier attacks. The same is true of diabetes, Bright's disease, and secondary syphilis. In persistent cough of doubtful origin careful examination should be made with reference to the existence of one or the other of these disorders. Gouty bronchitis may supervene suddenly, with amelioration of other symptoms of that affection, and in all these diseases, which are attended by the presence of toxic elements in the blood, and often pursue an insidious course, acute bronchitis may be the earliest symptom for which the

patient consults a physician. The importance of urinary analysis is obvious.

The influence of cardiac lesions in promoting bronchitis must be borne in mind, and in some cases of thoracic aneurysm hoarseness and cough are prominent symptoms due both to bronchial catarrh and to pressure on the recurrent laryngeal nerve.

The diagnosis further relates to the various local and subjective symptoms which have been previously described and to the physical signs. As a rule, in simple bronchitis there is no great elevation of temperature. Rigors seldom occur. Cough is more urgent than in pleurisy, less painful than in pneumonia, while the dyspnœa is seldom so marked as in either of these affections, except when the bronchial inflammation extends to the smaller tubes.

The expectoration, which is not a prominent feature in pleurisy, presents positive characteristics in bronchitis, to which allusion has already been made (page 127), and it is devoid of the rusty color and adhesive quality so noticeable in pneumonia. It is to be observed, however, that the latency of the cough and the absence of expectoration in young children and in feeble old persons who are suffering from pneumonia, with the difficulty in eliciting physical signs, may render the exclusion of that disease, for a time at least, impossible.

The bacteriological examination also serves to identify the pneumonic and phthisical sputa, although the specific bacilli of tuberculosis are found with difficulty in the earliest stages of that disease. In such cases, as in the acute miliary form of tuberculosis, the evidence derived from the sputa and the physical examination may be negative. The differential diagnosis must then be based upon the history, the cachectic appearance, the high irregular fever, and the constitutional symptoms which distinguish these conditions from bronchitis. Hæmoptysis, which does not belong to the history of bronchitis, is strong evidence of tubercular disease and may be an early symptom.

The physical signs of acute bronchitis may be positive or almost entirely negative. The percussion tone in general is normal, but dulness may be sometimes detected over areas where there is collapsed lung.

The respiratory murmur, as stated above, is normal in quality, but the expiratory sound may be somewhat prolonged and is of low pitch. When the finer bronchi are invaded subcrepitant râles are heard with inspiration and expiration on both sides, especially toward the bases. The bilateral distribution of the râles and the absence of bronchial breathing, dull percussion tone, bronchophony, and increased vocal fremitus serve to distinguish bronchitis from pneumonia, and, as a general thing, from broncho-pneumonia also.

Prognosis.—Although simple acute bronchitis is seldom fatal in healthy adults or children, still, bronchitis in general is an important factor in the death-rate of cities, especially in northern latitudes. The proportion of deaths from this cause to the total mortality may rise, in certain years, as high as 10 per cent., or even higher in particular localities. But 4 or 5 per cent. of the total mortality must be regarded as high, except when some unusual epidemic influence prevails. The ratio of deaths in midsummer to those in midwinter is about as one to four,

and at all seasons the victims are chiefly among debilitated individuals at both extremes of life, in whom the tendency is to asthenia and extensive involvement of the smaller bronchi.

In the milder forms of acute bronchitis, in which the catarrh affects the larger tubes only, recovery takes place in a few days or it may be in a week or two. But the disease is often protracted by exacerbations for several weeks through negligence or extreme susceptibility to external influences. It may result in chronic bronchitis.

In adults even, when the finer tubes in both lungs are extensively invaded and much cyanosis and dyspnœa is present, there is danger to life. Unfavorable signs are difficult expectoration and obstruction in the bronchi, rapid, shallow breathing, feeble pulse, delirium, and marked systemic depression, with a low temperature.

Acute bronchitis, when it is symptomatic in infectious diseases, runs a variable course and is sometimes dangerous. Thus, in measles the catarrhal process may be violent, with extensive pulmonary congestion and œdema, to which the patient succumbs. But in this disease, as in diphtheria, when cases are fatal from pulmonary complications the inflammation has often extended also to the bronchioles and alveoli. The danger from the bronchitis of measles is confined mostly to young children. An analysis by the writer[1] of 366 cases of measles, 69 only being under ten years of age, showed but 5 deaths. One case only was fatal from bronchitis, and in but few others did this feature of the disease excite apprehension.

In some cases of typhoid fever the brunt of the disease at an early stage falls upon the pulmonary system, and the bronchitis is the source of much discomfort, sometimes of danger. Thus, in 676 cases admitted to the Boston City Hospital for typhoid fever,[2] bronchitis was noted as a severe symptom in 74 (11 per cent.), and in 2 cases this complication was the principal cause of death. The bronchitis of typhoid, however, is not, as a rule, of serious importance.

An attack of acute bronchitis when associated with organic disease of the lungs or heart, especially if there is emphysema and right ventricular dilatation, is often a source of grave anxiety as to the immediate prognosis; and in all toxæmic affections, such as gout, rheumatism, Bright's disease, and diabetes, acute bronchial catarrh must be considered a very undesirable complication. In diabetes the pulmonary symptoms may be sudden and urgent, with more or less stupor—an index of deep systemic poisoning.

TREATMENT.—The treatment of acute bronchitis must be adapted to the age of the patient, the period of the disease, and to its nature, whether primary or secondary. In the latter case the underlying malady or cachexia must be recognized and treated as well as its bronchial manifestation, and under all circumstances the therapeutic measures which are adopted should be largely symptomatic and general, directed to the individual patient and his condition, with a judicious use of such specific remedies as have been found best to meet the different stages of the catarrhal inflammation as they arise. In a large proportion of mild cases the disease passes off in a few days with no medicinal treatment

[1] *Medical and Surgical Reports of the Boston City Hospital*, fourth series, 1889.
[2] *Boston Medical and Surgical Journal*, April 7 and 14, 1892.

whatever. Under this category would probably come most of the cases which are supposed to be aborted by a full dose of some drug to which special virtue is attributed—sulphate of quinine, for instance, since this salt is quite ineffectual in checking the course of bronchitis of ordinary severity. It is certain, however, that a cathartic, a hot mustard foot-bath, a diaphoretic draught, and a good night's sleep are of material service in aiding the system to throw off an incipient catarrhal attack under favorable individual conditions.

It has not been heretofore supposed that any drug possessed the property of cutting short a well-developed attack of bronchitis. The statement by Grün before referred to, that this disease is associated with the presence of a specific bacillus, was followed by experiments to discover an agent which would stop the growth of this organism. Acetanilid was said to possess this property, and the same author further states that the administration of this drug in 25 cases of acute catarrhal bronchitis was attended by uniformly favorable results, in that the disease was always arrested in a few hours. The dose administered was 5 grains every other hour. These statements require confirmation by clinical experiments on a large scale. Negative results were obtained by the writer in a few cases. In one there was prolonged cyanosis.

At the onset of acute bronchitis in its severer forms the treatment is to be directed to the general condition and surroundings of the patient and to the relief of symptoms. A warm, sunny room is desirable, and, while the cough is still dry and there is respiratory oppression, much relief may be afforded by charging the atmosphere with steam from a kettle or by frequent inhalations from the steam atomizer; or the vapor from a pitcher of boiling water, to which has been added a drachm of the compound tincture of benzoin, may be inhaled. When a room is charged with steam, however, proper ventilation must be maintained, as the heat is often excessive.

The bowels should be opened by a mild mercurial, gray powder, 10 grains, calomel, 3 to 6 grains, or blue pill, 5 grains, followed by a saline laxative. Free action of the skin is to be promoted by sweet spirits of nitre or some other simple diaphoretic, and the disturbed gastric function is to be considered in the avoidance of too much feeding. This point is often overlooked in children until the loaded stomach relieves itself by acid vomiting.

Emetics are useful in children who cannot expectorate, but usually at a later stage when the secretion is abundant and there is oppression from filling up of the bronchi. Of this class of drugs ipecacuanha is the best, in the form of the wine or the powder, and its action may be hastened by draughts of warm water or mustard and water or by tickling the fauces. The dose of the wine is a teaspoonful or two, repeated in fifteen minutes if necessary; of the powder, 5 grains to 10 grains. In urgent cases a subcutaneous injection of apomorphia is indicated, $\frac{1}{100}$ to $\frac{1}{10}$ grain, but the depressing effect of full doses of this drug is to be borne in mind. This objection applies more strongly to the use of preparations of antimony in feeble subjects, even in small, sedative doses. If tartar emetic is used in the early part of an attack, a quarter of a grain every three hours or from fifteen to twenty minims of anti-

monial wine is a sufficient dose for an adult. Citrate of potash in drachm doses in solution may be added.

The medicinal treatment further relates to the use of expectorants, sedatives, and narcotics symptomatically, the mode of their administration varying in accordance with the urgency of the cough and the quantity and quality of the secretion. The syrup of squills and of ipecac may be given in frequent half-drachm doses, or the wine of ipecac, in small quantity, 10 to 20 minims, unless nausea is produced. As the natural tendency to recovery is so strong, overdosing should be guarded against, and in this form of bronchitis it is seldom that at a later stage the more stimulating expectorants, such as the carbonate and chloride of ammonium or senega, are needed. While the free use of opiates is contraindicated in cases with much congestion and a tight cough, still there is no remedy which takes their place if proper caution is used. A few drops of liquid Dover's powder or small doses of codeine, ¼ grain, may be given to most patients every three or four hours for the relief of the harassing cough and insomnia from which they so often suffer, and frequently a full dose of morphine is the very best remedy.

The following combinations have proved useful:

℞. Syrupi scillæ,
Tincturæ opii camphoratæ,
Syrupi tolutanus, āā. ℥j.—M.
Sig. Teaspoonful every three hours.

℞. Tincturæ opii camphoratæ,
Vini ipecacuanhæ, āā. ℥ss;
Spiritus ætheris nitrosi, ℥ij;
Glycerini, ℥j;
Aquam destillatam, ad ℥vj.—M.
Sig. Teaspoonful or two every three hours, according to age.

℞. Syrupi scillæ,
Syrupi ipecacuanhæ,
Extracti pruni Virginianæ, fl., āā. ℥j.—M.
Sig. Teaspoonful every three hours; expectorant.

Chloral, 5 to 10 grains, the bromides, dilute hydrocyanic acid, 1 or 2 drops, and the various hypnotic drugs are more or less serviceable in allaying spasmodic cough. Chloroform water in drachm doses every two or three hours is also a good sedative. Hyoscyamus and conium, belladonna and lobelia, stramonium and cannabis indica, are among the other drugs which are used in this complaint for their narcotic or sedative effects, but no special directions can be given for the employment of one rather than another, except the general caution against the use of strong remedies when milder ones will answer the purpose.

The value of local applications must not be overlooked. Hot fomentations to the chest promote comfort at the beginning of the attack. The rubefacient effect of turpentine or of weak sinapisms tends, through the determination of blood to the surface, to relieve the internal congestion. Sometimes the support of a tight swathe is beneficial. Blood-

letting and blisters are seldom called for, but dry cups may be of advantage, or friction of the chest with a stimulating liniment, the linimentum ammoniæ or the linimentum saponis. All external irritants must be used with care in children.

There are certain cautions to be observed in the treatment of acute bronchitis in infants and in the aged, and these relate chiefly to the latency of the symptoms and the danger of filling up of the bronchi and extension of the process to the bronchioles; to the more frequent necessity for emetics in the very young and for stimulating expectorants and alcohol in the old; and to the intolerance of opiates and of depressing drugs at both extremes of life. The inhalation of oxygen has been strongly recommended for this class of patients.

A simple and restricted diet during the earlier stages of the disease should be followed during convalescence by abundant food, tonics if needed, such as iron, quinine, and malt, and a return to fresh air and exercise as soon as is consistent with due care to prevent a recurrence of the malady.

In the prophylaxis must be included warm clothing, dry feet, and avoidance of exposure, but it is highly important for the young to become inured by a regulated outdoor life to the ordinary climatic changes, or, if this cannot be effected, migration for one or more seasons to a warmer latitude may be essential. It is well to become accustomed to daily bathing with cool or cold water, followed by friction with a hair mitten or coarse towel, unless the circulation proves to be very deficient and there is prolonged chilliness after the bath.

In regard to the numerous cases of acute bronchitis which are secondary to other diseases, their treatment must be that which is appropriate to the primary affections, combined with symptomatic remedies for the bronchial complication. Thus, in valvular lesions of the heart bronchitis is usually a sign of failing compensation and is often accompanied by pulmonary œdema and hæmoptysis. Treatment by rest, digitalis, 10 drops of the tincture t. d., and other cardiac stimulants is indicated. Gouty bronchitis, which often tends to the subacute or chronic type, may assume a very acute congestive form, with laryngeal spasm and œdema of the glottis very threatening in character. Such cases occur in plethoric subjects, and are best treated by rapid depletion with hydragogues (elaterium, $\frac{1}{6}$ grain; croton oil, 1 drop), or even by venesection.

The acute bronchitis of influenza is largely of nervous origin, and often is not amenable to the ordinary catarrhal remedies. The severe exacerbations of bronchitis which occur in diabetic patients are probably due to the accumulation of toxic products in the small intestines—acetone and diacetic acid—and call for prompt elimination by cathartics and the rapid administration of alkaline carbonates. It is not advisable to try to diminish the urinary secretion, but after free evacuations, if the cough is urgent and stupor is not a prominent symptom, codeine may be given cautiously, in doses of $\frac{1}{4}$ to $\frac{1}{2}$ grain.

CHRONIC BRONCHITIS.

DEFINITION.—A chronic catarrh of the bronchial mucous membrane, in uncomplicated cases usually the result of repeated attacks of acute bronchitis, but also an accompaniment of chronic affections of the lungs (tuberculosis, emphysema) and heart, and of such conditions or diseases as cause constant inflammation of the bronchial mucous membrane by an external or an autogenetic irritant (dust inhalation, gout, alcoholism).

PATHOLOGY.—In those cases which develop from acute or repeated subacute attacks the catarrhal alterations are for a time the most prominent characteristics. The blood supply remains abnormally abundant, and the extrusion of lymph and white corpuscles from the vessels continues. Gradually, however, productive changes set in and a true hypertrophy occurs. As a rule, no very marked changes appear in the epithelium at first beyond an infiltration with round cells, although in some cases there is a decided hyperplasia. The bloodvessels increase in number, and extensive infiltration of the submucous and intermuscular connective tissue with leucocytes takes place. The most marked hypertrophic changes occur, however, in the elastic tissue and in the muscular layer. The increase in the muscles is probably the result both of inflammatory irritation and increased function. The mucous glands become distended and their acini filled with muco-serous exudation. Although the adventitia and cartilages are at first not involved, the process finally extends to them also. The inflammation in the adventitia is a productive one, and in time extends to the interlobular connective tissue. The cartilages undergo fatty disintegration or fibrous transformation. As the result of these changes the mucous membrane appears reddened and thickened, often velvety, from the papillary development of the bloodvessels. The hypertrophy of the muscular and elastic tissues often results in the formation of bands which cause more or less stenosis of the bronchi and subsequent bronchiectasis and emphysema.

As time goes on, however, these conditions give place to progressive fibroid atrophy. The epithelium subsists with difficulty and disappears to a greater or less extent. The muscularis undergoes fatty degeneration or fibrous change occurs. In some cases, however, the walls become infiltrated with lime salts. The glandular tissues atrophy. The new tissue thus formed is weak and yielding, and little calculated to withstand the increased air pressure from cough and stenosis. Hence bronchiectasis follows, and the low vitality of the tissue and the presence of decomposing secretions often lead to ulceration of the walls. The pathological changes occurring as the result of pulmonary venous stasis from any cause, as in heart disease, differ decidedly from those in chronic catarrhal bronchitis, although this latter condition is often superadded.

SPUTUM.—The expectoration varies from almost none in the "catarrhe sec" to one or two quarts daily in the "serous bronchorrhœa." In this form it is thin, frothy, and purely serous. Ordinarily, however, the sputum is more or less purulent and contains yellowish or yellowish green masses. In putrid bronchitis it is thick and greenish brown, with a repulsive, sweetish odor. It is abundant, and tends to separate into three layers, the upper consisting of frothy mucus with

more or less shreds extending down into the middle layer, which is thin and watery. The lowest is composed almost entirely of pus, in which dirty, yellowish gray masses, the so-called "Dittrich's plugs," are often present. These are composed of decomposed pus corpuscles, detritus, and bacteria, and often contain crystals of the fat acids. Lumniczer[1] isolated several varieties of bacteria from the sputum of fetid bronchitis, among them the pus cocci. He also found a fungus which, when grown on agar, emitted the characteristic odor of putrid bronchitis. When introduced into the lungs and bronchi of rabbits it produced a local inflammation.

Etiology.—Chronic bronchitis is usually an affection of middle age or advancing years, and is rarely seen in young subjects. It results in most cases from the frequent occurrence of acute attacks, and therefore all the causes which have been enumerated in connection with acute bronchitis are also concerned in the etiology of the chronic form.

Sometimes the disease may assume a chronic type from the beginning; or, rather, in persons who have not been subject to acute bronchitis a subacute attack may gradually merge into a chronic catarrh. This occurs mostly in elderly people, in whom the cough is secondary to some constitutional cachexia or condition, such as gout, heart disease, or alcoholism. While repeated attacks of catarrhal inflammation of the bronchi usually precede the chronic disease, the development of this troublesome affection is not a very common result of simple primary bronchitis, however often it may recur. Chronic bronchitis is often symptomatic. It accompanies phthisis, emphysema, and asthma, although it may be regarded as the cause rather than the effect of the vesicular dilatation. Chronic cardiac lesions, especially mitral stenosis and incompetency, render their subjects liable to pulmonary congestions and persistent cough.

The constant presence of toxic elements in the blood, which seek elimination through the bronchi, must be regarded as a prominent factor in the etiology, although many gouty, rheumatic, and diabetic subjects, through reasonable care in their modes of life, escape with occasional acute attacks. A subacute or chronic form of bronchitis is very common in persons who drink much wine or spirits, and is often associated with more or less gastric catarrh. The inhalation of various irritating substances, previously mentioned, tends to establish a permanent bronchial inflammation, and in cold, damp climates individuals with sensitive mucous membranes may have a constant winter cough which eventually becomes chronic.

Symptoms.—The development of chronic bronchitis is, in most cases, gradual, extending over a period of years, during which there may be many acute or subacute attacks before the disease is firmly established, in accordance with the varying activity of the predisposing influences. The symptoms are most marked in cold weather, and may abate, to a considerable degree at least, in summer. They are in the main like those described as pertaining to acute bronchitis with certain characteristic aggravations. These relate chiefly to the nature of the cough, the quantity and quality of the expectoration, and the systemic exhaustion which attends the disease in old and feeble persons. Noticeable sub-

[1] *Wiener med. Presse*, 1888, xix. 666.

jective symptoms are dyspnœa and a certain degree of pain or oppression under the sternum, which however, may not cause positive discomfort except after considerable exertion.

Cough, which in the milder forms of chronic bronchitis is neither severe nor constant, and at times almost disappears, gradually becomes more urgent, perhaps paroxysmal, in character. As the disease goes on this symptom is provoked by slighter causes, with shorter periods of intervening relief, until in the worst cases the cough becomes violent and exhausting. It is often very troublesome at night, and the resulting insomnia may be followed by morning paroxysms which are only relieved by free expectoration. The expectoration presents the varying characteristics which have been before mentioned. In ordinary cases it is moderate in quantity, muco-purulent, and easily raised.

In "dry catarrh" the smaller tubes are often involved; there is a scanty secretion, tenacious or bloodstreaked, sometimes in small pearly masses which are expectorated with difficulty after long spasms of coughing. This form of catarrh, usually due to asthma or some toxæmic state, such as gout or alcoholism, frequently leads to emphysema. In the congestive bronchitis which is incident to organic disease of the heart bloodstained sputa and hemorrhage are often seen.

In certain other cases there is bronchorrhœa. This occurs chiefly in old persons or in those with bronchitis of long standing, who may raise several pints of sero-purulent or muco-purulent fluid daily. Expectoration of this large amount is not always attended by much discomfort, but the cough is usually in prolonged and exhaustive paroxysms. Fetid expectoration, suggestive of pulmonary gangrene or of empyema discharging through the bronchi, may be an accompaniment of bronchitis with dilated bronchi and retained sputa. Occasionally there is no special change in the tubes to account for the fœtor.

As chronic bronchitis is so often secondary to some other affection, its symptomatology and course must be largely dependent in many instances upon the stage and progress of the primary disease. In the milder cases there is little or no fever, the general condition remains good, and life is not endangered; whereas in the severer forms, especially in aged persons, either directly or indirectly this disease adds an important element of gravity to the prognosis.

Physical Signs.—The physical signs of chronic bronchitis, *per se*, vary little from those described in connection with acute bronchitis. The percussion tone is unchanged, except when it is exaggerated by emphysema or modified over certain areas by the occlusion of bronchi. The respiratory murmur, as a rule, is of normal quality, but often of diminished intensity, and the expiratory sound may be noticeably prolonged. Occasionally a harsh quality is observed. There are no characteristic changes in the vocal resonance or fremitus. Dry and moist râles, coarse and fine, varying in site from time to time, may be heard over both sides of the chest. In many cases with pulmonary emphysema the contour of the thorax is changed, there is less expansion and vibration, and the percussion tone is hyper-resonant throughout. The cardiac dulness is then obliterated or diminished in area by the overlapping lung, and the heart, diaphragm, and subjacent organs may be depressed.

Cardiac dilatation, which is often present, cannot always be determined by physical examination.

Diagnosis.—Chronic bronchitis in its simple, uncomplicated form is easy of diagnosis, both from the previous history and the positive symptoms and signs attending its course, as well as from the absence of those features which serve to distinguish pneumonia, phthisis, and other pulmonary affections. The bilateral distribution of the physical signs without evidence of lobular or lobar consolidation, the moderate degree of fever and constitutional disturbance, and the slight falling off in weight, color, and general condition are usually sufficient to determine the presence of bronchitis rather than either of the other diseases mentioned.

Strong negative evidence is afforded by the failure to discover the tubercle bacilli in the sputa. This is especially valuable in the severer forms accompanied by bronchiectasis, in which there may be a considerable degree of hectic fever and emaciation with doubtful physical signs. The detection of the primary disease, condition, or cachexia upon which the bronchitis depends is all-important both for prognosis and treatment. In questionable cases this involves careful inquiry into the state of the various organs and functions, respiratory, cardiac, renal, and digestive, and into the occupation, mode of life, and habits of the patient.

Complications and Sequelæ.—There is a marked tendency in the subjects of chronic bronchitis to acute exacerbations, in which the disease may extend to the smaller tubes, and through their obstruction collapse of portions of the lungs may take place. Broncho-pneumonia may also result from further extension to the capillary bronchi. But the most frequent sequels of long-standing bronchitis are a general distention of the air vesicles (pulmonary emphysema) and permanent dilatation of the bronchi (bronchiectasis). The coexistence of asthma contributes to these results. Enfeebled action of the heart through dilatation of its right cavities often ensues, with consequent pulmonary engorgement, congestion of the liver and other organs, and finally œdema of the extremities. It cannot be said that chronic bronchitis causes a strong predisposition to the development of pulmonary tuberculosis, but the presence of bronchiectatic cavities or of interstitial fibroid changes, as in cases due to certain dusty occupations, may tend to promote invasion of the lungs by the tubercular germs.

Prognosis.—There is little immediate danger to life from chronic bronchitis in itself, although it may indirectly cause death from its aggravating effect upon the primary disorders of which it is symptomatic or from the complications that it causes. Thus, in chronic cardiac disease with accompanying bronchitis the already laboring heart is further weakened and dilated by the strain of constant coughing. Hæmoptysis sometimes is a relief to the pulmonary congestion present in such cases. The direct results of prolonged bronchitis, which have already been described—viz. emphysema of the lungs, bronchiectasis, and cardiac dilatation—while serious and disabling to a greater or less degree, may be compatible with a certain amount of comfort and even of work. Eventually, however, in a considerable number of cases these secondary manifestations become more and more

troublesome, or acute exacerbations involving the finer tubes set in, and the patients succumb to extensive pulmonary congestion, broncho-pneumonia, or exhaustion. In feeble or aged persons the inability to free the tubes of mucus is a source of danger. Therefore this disease is not to be regarded as a slight one, since it is always incurable after the above-mentioned secondary changes in the bronchi have occurred, but much may be accomplished in the way of prophylaxis and treatment.

TREATMENT.—The treatment of chronic bronchitis should be both general and symptomatic. It must be directed in each individual case to the primary disease or cause upon which the catarrhal condition depends, if this is a secondary manifestation, or, if not, to the maintenance of the general health and the prevention of exacerbations, as well as to the relief of the more obvious symptoms by local or medicinal remedies.

Therefore the therapeutic measures to be advised are palliative or curative, more frequently the former, and they relate to the following different conditions:

1. Secondary or symptomatic bronchitis;
2. Winter cough and chronic bronchitis of moderate severity;
3. Chronic dry catarrh;
4. Bronchorrhœa;
5. Fetid bronchitis.

In many cases of organic disease or chronic cachexia, when bronchitis is merely an incident in their course, palliation only is to be expected, and even this is not always possible. This is true of the congestive cough which may occur in advanced cardiac disease, although it often happens that compensatory action is partially restored and the pulmonary symptoms are relieved by rest in bed, combined with the use of salines, diuretics, and stimulants. Exhausting catharsis is to be avoided in such cases, and much benefit may result from strophanthus, digitalis, and the moderate use of wine or spirits. Nitro-glycerin in $\frac{1}{100}$ grain doses, and the nitrites, given with a view to diverting blood to the periphery, are sometimes of service.

For the harassing cough caused by the pressure of aneurysms or other intra-thoracic tumors, especially those which involve the pneumogastric nerve or its branches, vascular sedatives and opiates are the only palliatives. Iodide of potassium is often used.

A large proportion of the cases of chronic bronchitis may be traced to the rheumatic and gouty diatheses and to alcoholic excess, and can be relieved or cured only by measures directed in the main toward these conditions. The tendency of rheumatic subjects to develop bronchitis which gradually becomes chronic is very noticeable, and is to be met by extreme care in the prevention of exposure during cold weather and during the exacerbations which often occur by the administration of the salicylate of sodium, 5 grains hourly, alkalies, and other antirheumatic remedies. The smaller tubes not infrequently suffer; broncho-pneumonia may result; therefore bronchitis must be regarded as one of the chief dangers to this class of patients.

This danger is still more pronounced in the gouty, and, as bronchitis of a subacute or chronic type often appears in persons of this diathesis who do not present the symptoms of acute gout, not infrequently in

those of the female sex, the causal relations must be sought for and considered in the treatment. The chief indications are to prevent the accumulation of gouty poison in the system, and in case of bronchial exacerbations to promote its rapid elimination. The use of alkalies, Carlsbad salts, and laxatives, combined, if necessary, with colchicum (10 to 20 drops of the wine of colchicum root, thrice daily), is usually sufficient to relieve the milder forms of gouty cough, but when, after indiscretions in drink or diet, severe paroxysms of bronchial and laryngeal catarrh supervene, the condition may become alarming. Previous attacks have often brought about some degree of emphysema. Dyspnœa is marked and there is a dusky color, with intense congestion of the bronchial mucous membrane and a dry cough. Severe spasmodic croup may occur. Hot pediluvia and rapid depleting measures by evacuating enemata and prompt catharsis are here indicated. The abstraction of blood by leeches or cups may be resorted to. Opiates are contraindicated in this condition, but if the spasmodic dyspnœa is extreme it may be relieved by a few whiffs of ether. Vascular sedatives, such as aconite in small repeated doses, 2–5 drops of tincture of the root, tend to allay the bronchial irritation, and in favorable cases the scanty, viscid, bloodstreaked sputa give place in a few days to freer secretion of a muco-purulent or nummular character, which is more easily expelled by the aid of an expectorant, although drugs of this class are of little service at the outset. In such cases there is often some degree of arterio-sclerosis, with an acid, albuminous urine, usually of high specific gravity.

The alterative action of calomel in small doses, $\frac{1}{10}$ grain every two hours, is beneficial in gouty bronchitis, but mercurials must be administered with caution when a large quantity of urine of low specific gravity, with a trace of albumin, gives warning of those interstitial changes in the kidney which are so common in gouty subjects. Salivation may follow if they are freely used. The supervention of an acute attack of gout or of an eczematous eruption upon the suppression of gouty bronchitis in certain individuals suggests that palliation of the catarrhal symptoms may be better than their cure, even if this could be attained.

Prolonged attacks of bronchitis in diabetics demand special care in the prevention of the acute exacerbations to which previous allusion has been made.

Syphilitic, strumous, and rachitic bronchitis are to be treated by those specific and constitutional measures which are applicable to the diseases in question. So also is the cough which may attend the development and progress of malaria.

The injurious effects produced by the constant inhalation of vapors or dust, common among workers in chemicals and in mills of various kinds, can only be obviated by the constant use of respirators or a change of occupation. The former method is usually found impracticable, because it cannot be enforced.

In the prolonged catarrhal inflammation which may attend the presence of tuberculosis, pleural adhesions, hepatic diseases, and other more distant organic and functional disorders due regard must be paid to the

secondary nature of the bronchitis in the measures which are adopted for its relief.

The treatment of "dry catarrh," the "catarrhe sec" of Laennec, cannot be disassociated from that which is appropriate to many of the conditions previously described, as this form of bronchitis is most frequently observed in rheumatic, gouty, and alcoholic subjects or as an accompaniment of asthma and emphysema. Its essential features being a hard, dry, paroxysmal cough and scanty secretion, due to irritating bronchial congestion, the indications are to relieve the system of the toxic elements on which the bronchitis depends, to attend carefully to the functions and to the general condition of the patient, and to allay the subjective symptoms as they present themselves. Of these cough is the most prominent, and for its relief sedatives, narcotics, and expectorants are often necessary, the effects of these drugs varying greatly in different cases according to the susceptibilities of the individual and the nature of the exciting cause. If there is much fever and oppression during an exacerbation, opium is not a good remedy, and under any circumstances the constant use of this drug is objectionable.

There are some cases of dry catarrh, however, which are better controlled by opiates than in any other way, either in full doses of morphine, $\frac{1}{8}$ to $\frac{1}{4}$ grain, codeine, $\frac{1}{4}$ to $\frac{1}{2}$ grain, or paregoric, or in combination with ipecacuanha and other expectorants, as in the form of Dover's powder in repeated small doses of 3–5 grains. Dilute hydrocyanic acid, asafœtida, and the bromides may be tried in cases with a decided nervous element; and narcotics, such as hyoscyamus, conium, belladonna, and stramonium, are often recommended. Chloral hydrate and butyl-chloral in 5-grain doses are useful for their sedative and hypnotic influence, as are sulphonal and trional, 10–20 grains, paraldehyde, and other drugs of this class. But the depressing effect of all these remedies must not be overlooked, and for the same reason the nauseating expectorants are seldom desirable. Iodide of potassium, in 5-grain doses gradually increased, may be very effectual in loosening the bronchial secretion, but often it is useless, and in some cases, through lack of tolerance, it aggravates the catarrhal symptoms. Chlorate of potash also aids expectoration. A moist atmosphere saturated with steam facilitates breathing, and the inhalation of a soothing vapor or spray is beneficial.

The treatment of the ordinary forms of chronic bronchitis which are not dependent upon any primary disease or cachexia, but which are simply the expression of individual susceptibility to catarrhal inflammation from immediate exciting causes often repeated, must be in the main symptomatic, varying in accordance with the stage of the malady, the age of the patient, and the degree of bronchial dilatation and emphysema which may have occurred.

With regard to the many cases of simple "winter cough" which are included in this category the prophylactic and climatic measures which naturally suggest themselves as of chief importance will be considered later in connection with the general management of all forms of chronic bronchitis.

While it is true that a routine or empirical mode of drugging may be harmful, and that the medicines prescribed for bronchitis are often of little or no service, still the judicious use of narcotics, expectorants,

stimulants, inhalations, or tonics will prove beneficial in a large proportion of cases. Previous reference has been made to the various narcotic and sedative drugs, and they are always indicated when painful, exhausting cough, with a nervous or spasmodic element, is out of proportion to the amount of bronchial secretion which requires expulsion. Many combinations of morphine, codeine, and opium with the milder expectorants, such as squills and ipecacuanha, are of traditional value in such cases.

Thus:

℞. Morphinæ sulphatis, gr. ij;
Syrupi scillæ,
Syrupi pruni Virginianæ, āā. ℥ij.—M.
Sig. Teaspoonful every three hours.

℞. Codeinæ, gr. iv;
Acidi hydrocyanici diluti, ♏xxx;
Syrupi scillæ, ℥iv.—M.
Sig. Teaspoonful every four hours.

When, however, there is an excess of secretion and the cough is insufficient in expulsive force, then the more stimulating expectorants may be of much service if the patient is not too feeble to respond to their action. In this class are senega, in the simple syrup (℥j) or in the compound syrup of squills, U. S. P., 30 drops; the carbonate and chloride of ammonium, balsam of copaiba, and the terebinthinates, as in—

℞. Ammonii carbonatis, ʒij;
Aquæ destillatæ, ℥iij.
Fiat Solutio.
Sig. Teaspoonful in wineglass of water every three hours.

℞. Ammonii chloridi,
Extracti glycyrrhizæ, āā. ʒij;
Aquam, ad ℥iv.
Sig. Teaspoonful every three hours.

Cubebs is an ingredient of many cough lozenges. If antimonials, pilocarpine, or apomorphine are used as expectorants, they should be in small doses, and their effects must be carefully watched. The Calabar bean, from its stimulating influence on the pneumogastric, has been thought useful in chronic bronchitis with atonic dyspnœa, in doses of $\frac{1}{200}$ grain of eserine, cautiously repeated.

The following are good formulæ:

℞. Vini antimonii, ℥ss;
Liquoris ammonii acetatis, ℥iiss;
Syrupi aurantii,
Aquæ, āā. ℥iss.—M.
Sig. For dry cough. Tablespoonful in water every three hours.

℞. Vini antimonii, ʒij;
Spiritus ætheris nitrosi, ℥j;
Tincturæ opii deodoratæ, ʒij;
Aquæ, ℥ij.—M.
Sig. Dose, a teaspoonful every three hours.

℞. Morphinæ sulphatis, gr. ss;
Chloroformi, ♏xx;
Glycerini,
Aquæ, *āā.* ʒss.—M.
Sig. Sedative. Dose, a teaspoonful p. r. n. Shake.

℞. Chloroformi, ♏xxx;
Syrupi pruni Virginianæ, ʒij.—M.
Sig. Sedative. Dose, a teaspoonful every three hours. Shake.

℞. Morphinæ sulphatis, gr. iij;
Vini ipecacuanhæ,
Syrupi scillæ,
Syrupi senegæ, *āā.* ʒij.—M.
Sig. Expectorant. Teaspoonful every three hours.

℞. Ammonii carbonatis,
Ammonii chloridi, *āā.* ʒj;
Morphinæ sulphatis, gr. j;
Spiritus anisi, fʒss;
Syrupum pruni Virginianæ, ad ʒiij.—M.
Sig. Stimulating expectorant. Teaspoonful every three hours.

When there is excessive secretion (bronchorrhœa) there is also, as a general thing, bronchial dilatation or at least deficient power of expulsion, which must be met by remedies like those mentioned above or by others of an alterative character. Iodide of potassium, 5–30 grains t. d., alkalies, and the syrup of hydriodic acid, ʒj t. d., sometimes render the secretion more easily eliminable. Ammoniac is often used, alone or in the compound squill pill, U. S. P. Resort to emetics is seldom desirable. Certain cases improve under the use of tar (syrup. picis liq., ʒij–ʒiv t. d.), turpentine, terebene, or terpene hydrate, 5♏ in capsules t. d., but the choice of remedies must be largely experimental. Astringents given internally do not have much effect upon the bronchial mucous membrane, but inhalations of a stimulating, astringent, or antiseptic vapor or spray may be highly beneficial in diminishing the secretion. The steam atomizer affords a convenient means for the inhalation of medicated solutions, and in this way may be employed weak solutions of tincture of iodine, carbolic acid and creasote, tannic acid, alum, subsulphate of iron, and many other agents, which, by proper care in ensuring deep respiration, thus reach the bronchial passages.

In the same manner extreme irritability may be allayed by the inhalation of dilute solutions containing paregoric, morphine, cocaine, hyoscyamus, and other sedative drugs. Balsamic vapors, as that produced by the addition of a drachm or two of the compound tincture of benzoin to a quart of boiling water, sometimes have a favorable influence in reducing bronchorrhœa. But in old persons, when there is much exhaustion and inability to expectorate, little benefit results from measures directed to the bronchial system. General stimulating and sustaining treatment may bring partial relief, and occasionally oxygen inhalations give temporary comfort.

The odor in fetid bronchitis is best modified by the inhalation of carbolized fluids, creasote or menthol.

The following substances, in the amounts specified, may be added to 1 ounce of water for atomizing:

℞. Tincturæ iodii,	minims	2 to 10;
Acidi carbolici,	grains	2;
Creasoti,	minims	3;
Acidi tannici,	grains	2 to 10;
Alumini exsiccati,	"	3 to 15;
Liquoris ferri subsulphatis,	gtt.	5 to 20;
Tincturæ opii,	minims	5 to 30;
Tincturæ opii camphoratæ,	drachms	1 to 3;
Morphinæ sulphatis,	grain	½ to 1;
Sol. cocainæ hydrochlorici, 4 per cent.,	minims	30 to 60;
Tincturæ hyoscyami,	"	30 to 60;
Tincturæ stramonii,	"	30 to 60;
Tincturæ belladonnæ,	"	30 to 60.

Many other drugs may be used in the same manner or by vaporization.

In recent years the pneumatic cabinet has been much in vogue for the use of both condensed and rarefied air with the purpose of increasing expiratory force and aiding expectoration. This mode of treatment is applicable to cases of chronic bronchitis with retained secretions in which there is more or less emphysema. But emphysema of long duration is not, as a rule, much benefited. Successful results may also follow the use of the pneumatic cabinet in cases of persistent cough attributable to pleural adhesions.

Chronic bronchitis, being a disease which implies a certain lack of resistance on the part of the system, calls not only for the avoidance of depressing measures, but also, in a large proportion of cases, for the administration of stimulants and tonics. This is especially true in the aged and in persons with weak circulation and deficient power of expectoration. The moderate use of brandy, whiskey, or wine is available for most cases. Alcoholic subjects who still have fair recuperative energy are better with little or no stimulants, but when there is marked asthenia entire abstinence is often impracticable. The wine of coca is sometimes serviceable. Preparations containing iron, quinine, phosphorus, and strychnine are indicated, as well as the bitter tonics and infusions, for promoting appetite. When cod-liver oil is well borne it is a useful remedy for emaciation and debility. An emulsion of linseed oil may also be recommended.

Further than this, the careful regulation of the functions of the liver, bowels, kidneys, and skin is of the utmost importance. Laxative doses of blue pill, calomel, gray powder, or some other cathartic may be given at intervals. Many patients are more comfortable from the habitual use of saline aperients, such as the waters of Saratoga, Friedrichshall, Pullna, or Hunyadi Janos. When the urine is highly acid or loaded with lithates the waters of Carlsbad, Vichy, or Selters are indicated, and the various lithiated waters are also good diluents.

The functions of the skin are to be stimulated by bathing, cold, tepid,

or warm in accordance with the individual patient's reaction, and by friction with the coarse towel or mitten. Turkish baths must be used with some caution as to subsequent exposure.

Local Applications.—In chronic bronchitis, as in the acute form, the derivation of blood to the surface is of more or less service. When there is much congestion and a feeling of pulmonary constriction or oppression the prolonged application of weak sinapisms or of hot fomentations affords relief. Linseed meal poultices are much used, but they are heavy and often uncomfortable. The best guide to the continuance or abandonment of such measures is the degree of comfort or of annoyance which the patient experiences from their use. Stimulating liniments containing ammonia (linimentum ammoniæ), turpentine, camphor (linimentum saponis), or croton oil are often employed —croton oil less than formerly: the vesication which it causes is disagreeable. Dry cups are beneficial, and in sthenic cases with laborious breathing and cyanosis temporary relief may be obtained by bloodletting. This procedure, however, is now seldom resorted to except in grave emergencies in complicated cases, as chronic bronchitis is an affection in which, as a general thing, depletion is not advisable. Evident venous engorgement and dilatation of the right side of the heart are conditions which suggest the propriety of venesection.

PROPHYLAXIS.—One of the chief objects in the management of chronic bronchitis is the avoidance of exacerbations and the prevention or delay of those secondary changes in the respiratory apparatus to which previous reference has been made. Winter residence in a climate suitable to the individual is the best means of attaining these ends, but that is possible for the few only. Therefore the maintenance of a fair degree of comfort and capacity for work amid relatively unfavorable surroundings is the desideratum in most cases of this disease in its earlier stages. Care in regulating the diet, clothing, exercise, and atmospheric exposure will do much to make life comfortable. With a view to the general health a sufficient amount of simple, nutritious food should be taken at regular hours. The clothing, and especially the underclothing, should be warm enough to protect the patient thoroughly against climatic changes. In favorable weather outdoor exercise should be obtained, but not to the extent of producing over-fatigue or respiratory embarrassment. Caution must be observed in exposure to the cold, damp evening air, and in very sensitive persons, especially those at an advanced age, long periods of confinement to the house may be necessary. With proper ventilation, a sunny outlook, and congenial surroundings most old persons of domestic habits are far better in this way than if sent off in search of a climate. But the contrary is true of those subjects of chronic bronchitis who depend upon more or less outdoor life, and who have the means and vitality to seek and enjoy the benefits to be derived from a warmer latitude.

In a general way, it may be said that for chronic dry catarrh a warm, rather relaxing, atmosphere is best, while cases with profuse expectoration find more relief in a higher and drier climate. It is to be observed, however, that warmth is an essential element for most cases, and that high elevations like Colorado or the Engadine are unsuitable, especially when there is pulmonary emphysema. Places where there are sudden

changes of temperature, with high winds or dust, are also to be avoided. To particularize, among those equable and sedative climates which are good for the dry irritative bronchitis may be mentioned Bermuda, Nassau, or Florida. The inland southern portion of Florida has, for some cases, an advantage over the seaboard and the islands in its sandy soil and pine woods with little dampness. Southern California has many resorts which present the requisite attractions for the various forms of bronchitis.

The health resorts in the Carolinas and Georgia, such as Asheville, Aiken, and Thomasville, are much sought, but these places may be too cold in winter for very sensitive persons. The proximity of large numbers of consumptives has also a depressing effect.

On the other side of the Atlantic the Azores and Madeira offer that mildness, equability, and relaxation of climate which are characteristic also of Bermuda and other warm insular stations, and are soothing to the more irritative spasmodic forms of bronchial catarrh. Less equable and sedative, but sunny and more stimulating, are many points on the northern shore of the Mediterranean, especially along the Riviera, as at Cannes, Nice, and Mentone. These places, however, are not free from periods of cold, high winds and dust, as is true of Northern Italy. Those may suffer there who are dependent upon a high degree of warmth within the house, since fires and fuel are often insufficient for comfort. For this reason Southern Italy and the Mediterranean islands, as Capri, Sicily, and Malta, which are easily accessible, are more attractive to many invalids, as are the various resorts on the African side—Tangier, Algiers, the oasis of Biskra, Tunis, or Egypt. The ease with which travellers can now go direct from New York to Alexandria and the intervening Mediterranean ports takes many persons with chronic bronchitis to Cairo and the Nile, but it must be borne in mind that Egypt is dusty and that the lower river climate is by no means tropical. For all except the most delicate, however, the Nile voyage presents many attractions, but warm clothing is necessary everywhere in Northern Africa after the sun goes down.

During our summer and early autumn nothing is better in chronic bronchial ailments than a long sojourn in the balsamic woods of Maine, New Brunswick, or the Adirondacks for invalids whose condition and tastes enable them to enjoy the outdoor life in those regions.

PLASTIC BRONCHITIS.

Definition.—An acute or chronic inflammatory affection of the bronchial mucous membrane, in which extensive fibrinous coagula are deposited upon it, and finally expelled in paroxysms of dyspnœa and cough.

Under this disease must be included only those cases in which the fibrinous inflammation is primary in the bronchi. All those cases that are secondary to diphtheritic inflammation of the upper air passages must be excluded, as well as those due to extension from the lung alveoli, as in pneumonia and occasionally in phthisis. The only path-

ognomonic symptom is the characteristic sputum of moulds of a greater or lesser portion of the bronchial tree. Without their presence the disease cannot be diagnosticated during life.

The acute form is seen oftener in children and youths; the chronic in older people, especially at about the age of forty. The duration of the acute disease is from one to four weeks, of the chronic many years, even twenty-four [1] or twenty-five.[2]

Plastic bronchitis, from its rarity and the interest attaching even to single cases, has attracted the attention of many of the best authors. But no one writer has seen many cases, two or three being a large number.[3] Pathologists of wide experience have stated that they had never met with a case at the post-mortem table. It may happen that the records of large hospitals for a period of years contain no instance of this disease. But the literature of the past hundred years, not to mention earlier descriptions, contains many valuable contributions which give a clear picture of the different forms of this affection.

To refer briefly to some of the more important publications which have appeared during the present century, Cheyne [4] wrote on "Bronchial Polypus," a name previously given to this disease, and drew a distinction between the membranous casts without hæmoptysis and the solid, arborescent masses which attend or follow pulmonary hemorrhage, regarding the latter condition of far more serious import, as indicating probable organic disease of the lung. Further observations have shown the incorrectness of this deduction. In France, Andral [5] and in Great Britain, North,[6] Carswell,[7] Corrigan,[8] and Peacock [9] made further interesting contributions to this subject, Peacock giving a summary, the most complete at that time, of 34 cases in which fibrinous casts were thrown off from the bronchi, and drawing conclusions which have been confirmed by later observers. Watson's [10] graphic description of this disease is based upon the observation of 2 cases in brothers of middle age who were attacked within the same year, the disease being attended in both instances with hæmoptysis. Walshe,[11] Oppolzer,[12] Biermer,[13] and Lebert [14] in more recent years have added to the knowledge of this affection. Biermer's article is elaborated from 58 cases collected from all sources, 2 only having fallen under his own eye. Wilson Fox,[15] Douglas Powell,[16] and West [17] are among the latest writers on this subject. West's article presents an analysis of 52 cases collected from the literature of the preceding twenty years, and is supplementary to the researches of Lebert.

[1] Schnitzler, *Wien. med. Presse*, 1882, 828 u. 861.
[2] Kisch, *ibid.*, 1888, Bd. xxix. S. 33.
[3] Watson, 5 cases; Chvostek, 4 cases; Flint, 3 cases.
[4] *Edinburgh Medical and Surgical Journal*, vol. iv., 1808.
[5] *Clinique médicale*, t. iii., 1834. [6] *London Medical Gazette*, vol. xxii., 1838.
[7] Carswell, plates showing casts, London, 1838.
[8] Corrigan, *Dublin Med. Journal*, xvii., 1840.
[9] Peacock, *Med. Times and Gazette*, Dec., 1855, and *American Journal Med. Science*, April, 1855. [10] *Principles and Practice of Physic*, London, 1857.
[11] *Diseases of the Lungs*, London, 1860, p. 222.
[12] Oppolzer, *Vorlesungen*, Bd. i., 1868.
[13] *Virchow's Handbuch der Speciellen Path. u. Therapie.*
[14] *Deutsches Archiv für klin. Med.*, Bd. vi.
[15] *Diseases of the Lungs and Pleuræ*, Phila., 1892.
[16] *Diseases of the Lungs and Pleuræ*, London, 1893. [17] S. West, *Practitioner*, Aug., 1889.

In America, where this disease does not appear to have been at any time so common as in Northern Europe, the communications of Baumgarten[1] and of Austin Flint,[2] among others, describe this form of bronchitis as seen in this country, differing in no respect from the appearances as observed elsewhere. There are also original reports by Glasgow[3] and by Johnson and Davis to the American Medical Association[4] in later years.

Etiology.—From these various sources and from limited personal observations in New England it seems evident that in many of the subjects of plastic bronchitis there is a personal idiosyncrasy which determines the character of the fibrinous exudate without obvious reference to previous states of health or other existing conditions. The etiology is still in obscurity, owing to the scarcity of facts on which to base opinions. This affection is twice as common in men as in women, but in children the number is equally divided between the sexes. It occurs at all periods of life, but is more common between the ages of ten and forty. Hayn[5] reports a case in a newborn child, with autopsy. It may be more frequent in children than is generally supposed, as they swallow the sputum. Legendre[6] reports 1 case, and Fauvel[7] 5 cases, in children. This disease has seldom been observed in old persons. There are instances in which several members of a family have been attacked, and for this reason some writers attach importance to hereditary predisposition. Pichini[8] records 3 cases occurring simultaneously, as if due to some endemic influence. Cases are more numerous in the North than in the South, and occur most commonly in the spring. Chronic cases are generally preceded by a chronic bronchitis. Lehmann-Model[9] found an association with tuberculosis in his 7 cases, and many authors are inclined to consider this disease as an etiological factor. It is probably more correct, however, to consider it as coincident only. Syphilis, rachitis, alcoholism, pregnancy, typhoid fever, and various acute diseases have also been considered as causal, but presumably they have no more than a predisposing influence. The attacks in some cases seem to have been related to the menstrual period. Some of the acute cases have been considered due to an unusual localization of the Klebs-Löffler bacillus, but Escherich[10] inoculated animals with the membrane coughed up with negative results, and could not demonstrate the Klebs-Löffler bacillus in the bronchial secretions. Pichini[11] cultivated three varieties of micro-organisms from the expectoration in his cases, and injections of pure cultures into the tracheæ of animals are said by him to have caused a disease very similar to bronchial croup. In several instances

[1] Baumgarten, *St. Louis Med. and Surg. Journal*, Jan., 1869.
[2] Flint, *New York Medical Record*, Jan. 15, 1874; and *Diseases of the Respiratory Organs.*
[3] Glasgow, *Trans. Amer. Med. Assoc.*, 1879, and *St. Louis Courier of Medicine*, 1880—summary of American cases.
[4] *Journal American Med. Assoc.*, 1886–87.
[5] Hayn, *Caspar's Wochenschr.*, 1842, No. 51, S. 827.
[6] Legendre, *Récherch. sur quelques Malad. de l'Enfance*, Paris, 1846, 252, Obs. 7.
[7] Fauvel, *Clinique méd.*, iii. 225–251.
[8] Pichini, *Riv. clin. Arch. di Clin. Med.*, i. 105, 1889.
[9] Model, *Schmidt's Jahrb.*, 1890, Bd. 228, S. 250.
[10] Escherich, *Deutsch. med. Wochenschr.*, 1883, ix. No. 8.
[11] Pichini, *Riv. clin. Arch. di Clin. Med.*, i. 105, 1889.

the disease has followed thermic or chemical irritation (Lehmann-Model,[1] Pichini,[2] Pramberger,[3] Beschorner).[4]

England, Germany, and Switzerland are the countries in which plastic bronchitis has been oftenest observed, although it is by no means unknown in France, Italy, and the United States. Condition in life has little to do with its occurrence, as the poorer classes are no more subject to this affection than the rich. With more or less reason its onset in a large proportion of cases has been attributed to exposure in inclement weather.

PATHOLGICAL ANATOMY.—Anatomically, a distinction must be made between a diffuse and a circumscribed process. The former, reaching from the trachea to the finest bronchi, is found chiefly in the acute form. In chronic cases the disease is often limited to a single branch of the bronchial system. Autopsies have been rare in this affection, and

FIG. 15.

Bronchial casts, plastic bronchitis (from specimen in the Warren Museum, Harvard Medical School).

their results are considerably at variance. The most constant changes in the bronchi have been injection, reddening, and swelling of the mucous membrane—*i. e.* the general signs of inflammation. In some

[1] Model, *Schmidt's Jahrb.*, 1890, Bd. 228, S. 250.
[2] Pichini, *Riv. clin. Arch. di Clin. Med.*, i. 105, 1889.
[3] Pramberger, *Ueber fibrinöse Bronchitis*, Graz, 1881.
[4] Beschorner, *Klinische Vorträge*, N. F., 1894, 73.

cases a loss of epithelium has been demonstrated,[1] but it is usually intact. The croupous masses are sometimes adherent to the bronchial walls, sometimes free within the tubes, separated from the wall by secretion or by air. The exact method of origin of the casts is obscure, but the mechanism of production is probably the same as that of croupous membranes on other mucous surfaces—that is, a fibrinous exudation rather than a decidual epithelial proliferation.

Casts.—The casts as coughed up are rolled into bunches varying in size from a pea to a pigeon's egg, and imbedded in a milky, glassy, or bloody mucous menstruum. When spread out in water they are milk white or yellowish, and they form moulds of a greater or lesser extent of the bronchial tract. They are usually free from blood, but are

FIG. 16.

Bronchial tree, plastic bronchitis (from specimen in the Warren Museum, Harvard Medical School).

occasionally bloodstreaked. The ends often terminate in fine spirals, in which the Charcot-Leyden crystals are not infrequently found. These spirals probably originate in the bronchioles. The thickest parts of the casts are solid and concentrically formed; the medium-sized portions are often hollow and filled with air and mucus; while the terminal branches are solid and compact. Microscopically, the casts are composed of a hyaline, fine fibrous groundwork, in which masses of white

[1] Kretschky, *Wien. med. Wochenschr.*, 1873, 14–16.

corpuscles and occasional fat drops are imbedded. Blood globules are often present on the surface, but the epithelial cells of the air passages are much more numerous within. Chemically, they give the reactions for fibrin, resisting acetic acid. Beschorner,[1] however, has recently reported 2 cases, examined by Neelsen, in which they were composed mainly of mucin.

The size and length of these casts are subject to wide variations in different cases, those from the upper bronchi being relatively shorter than those from the lower portions of the bronchial tree. Three or four centimetres may be regarded as an average length, and ten or twelve the limit in recorded cases. They seldom reach the size of a pencil, and it is doubtful whether, when *in situ*, they fill up the entire bronchial lumen. When partially detached they may cause serious obstruction or occlusion at one or more points. Cases have been reported in which casts thrown off at brief intervals have presented, time after time, exactly the same length, size, and branching formation, down to the terminal bronchioles. They branch dichotomously, and have a concentric formation in layers corresponding to the gradual mode of exudation. In long-continued cases a hundred or more of these casts may be expelled. After rolling them out in a dish of water they may be preserved in a weak alcoholic solution.

Symptoms.—The symptomatology and course of the acute form will first be considered, although cases of this nature are much rarer than those of a more chronic type. Lebert, after careful search, found but 17 acute cases reported in the whole literature of the subject at that time (1869). During the next twenty years, among the 52 cases collected by West very few of the acute variety are recorded. In this form, which sometimes occurs in children, there may be slight prodromal symptoms as in an ordinary cold—fever, cough, and malaise. A chill sometimes ushers in the more acute manifestations, which, however, may come on abruptly without prodromata. In turn follow dyspnœa, cough, scanty expectoration, and severe substernal constriction. The dyspnœa grows urgent, with a sense of impending suffocation. The cough, at first dry and hard, soon becomes severe and paroxysmal, without, however, the hoarse tone of laryngeal croup. The sputa at an early stage show nothing characteristic, but they may contain a little blood, or even there may be considerable bronchial hemorrhage. Rarely fibrinous fragments are expelled during the early days of the disease, but in most cases the bronchial casts are thrown off at a later period, after several days of suffering, only by violent expulsive effort and often with free hæmoptysis. Relief follows at once. When recovery ensues the symptoms gradually abate, but in about one half the cases, so far as heretofore recorded, the signs of obstruction increase, cyanosis, stupor, and delirium ensue, and there is a fatal termination in a few days or life may be prolonged for two weeks. In such cases the fibrinous exudation may extend to the trachea, but the larynx is rarely involved. Walshe states that the disease in infants may run a favorable course in from ten to fourteen days and not recur.

The chronic form, on the other hand, oftener supervenes upon a more or less prolonged attack of bronchitis, although it may have a

[1] Beschorner, *Klinische Vorträge*, N. F., 1894, 73.

sudden onset as described above, with a less severe succession of symptoms than in the acute form. In most instances paroxysmal cough, distressing dyspnœa, and pulmonary oppression develop somewhat gradually, and these symptoms subside at once on the expulsion of fibrinous fragments or casts of a portion of the bronchial tree, to reappear again after a longer or shorter interval. Recurrent attacks may come on during weeks, months, or years, and their course is usually favorable, the tendency to this peculiar form of inflammation gradually disappearing. There is every grade of severity in the chronic type of plastic bronchitis, from a comparatively mild affection involving a small portion only of the bronchial tract, with more or less frequently recurring exacerbations during which an expulsion of membrane occurs, to the severer cases which approach the acute form in the intensity of dyspnœa, cough, and systemic disturbance, and in which extensive branching casts are thrown off. But recovery is the rule, although the general health may be greatly impaired by the frequency and severity of the successive attacks, which occur with no periodicity and with little regularity in their duration. In general these attacks last longer than in the acute form, sometimes a month or more, and during this time many successive casts of that part of the bronchial tract which is involved may be expelled. Cases are recorded in which casts exactly similar to each other in length, size, and branches have been coughed up day after day for a week. As in the acute process, hæmoptysis is also a very frequent incident in the chronic form, independently of any tubercular disease. An important symptom in the progress of the attack is the dyspnœa, which is sometimes constant, and relieved only by the expulsion of the obstructing plug through violent paroxysms of coughing. After this there is an interval of relative comfort, which may be but short lived if the fibrinous exudate is rapidly re-formed.

The signs of emphysema, atelectasis, or consolidation may be present, but the percussion tone is not changed by the plastic exudate in the bronchi alone. With large casts obstructing several bronchi the respiratory sound may be partially or completely cut off from portions of the lung, usually in the lower lobe, the normal murmur being restored as soon as the casts are expelled. There are no râles which are especially characteristic of this disease. The sonorous and sibilant râles of bronchitis are often heard. Flint regards the subcrepitant râle in a localized area, less diffused than in capillary bronchitis, as somewhat significant. Loud crowing, whistling, or flapping sounds have been described by several authors, doubtless due to the vibration around the casts which have become loosened in the bronchi. Diminished thoracic expansion and fremitus may be observed on the affected side, varying with the degree of obstruction which exists.

Complications and Sequelæ.—Irrespective of those cases in which antecedent or coincident tuberculosis or pneumonia may be present, the most frequent pulmonary change is compensatory emphysema, brought about by the supplementary action of those portions of the lung to which the air has free access and by the violent, paroxysmal cough. Atelectasis may occur if the affected bronchi are impervious. In chronic cases persistent bronchitis and emphysema may result, but tuberculosis of the lung cannot be regarded as a common sequel, although

in a considerable number of reported cases the patients have died of phthisis. In a few instances dropsy has supervened, probably secondary to emphysema, and pleurisy has been observed as a complication in several cases.

Diagnosis.—In so rare a disease it is very seldom that a first attack can be diagnosticated before the appearance of bronchial casts or fragments of the fibrinous exudate in the expectoration. When these are wanting, owing to the inability of the patient to cough them up, an inferential diagnosis only can be made from the physical signs and symptoms as previously described. When the disease comes on without a preliminary bronchitis, the localization of the signs in one side is suggestive of some affection other than a bronchial catarrh, which is commonly bilateral. In infants or young children the possibility of bronchial obstruction by a foreign body must be considered in estimating the physical signs and urgent symptoms. The absence of laryngitis and hoarseness serve to determine the bronchial origin in most cases where fragments of membrane are expectorated. After some cases of ordinary hæmoptysis decolorized clots moulded in the bronchi may be expelled, but the symptoms attending their expulsion are less severe than in plastic bronchitis, and their appearance is different from the casts described. The predisposition in old persons to bronchitis does not hold good in this form. Biermer found but 4 cases in persons over fifty, the oldest being seventy-two. In many cases of plastic bronchitis the paroxysms bear a strong resemblance to asthmatic seizures, and it is not improbable that the disease may thus be sometimes overlooked if the characteristic casts are not detected.

Prognosis.—Exclusive of those cases in which plastic bronchitis is associated with grave pulmonary disease, acute or chronic, the prognosis is relatively favorable. In many cases which have ended fatally the periodical expulsion of bronchial casts was a circumstance only in the course of pulmonary tuberculosis. It is not evident that the bronchial affection in itself was the cause of death in these cases. Bronchial casts which appear in the expectoration of acute pneumonia or diphtheria are not considered as related to the disease in question. In the few instances reported in which plastic bronchitis has occurred as a sequel to typhoid fever, scarlet fever, and pneumonia—that is, several weeks after these diseases—the course and duration of the attack appear to have been the same as in other cases. The coexistence of pemphigus, as observed in several cases, adds gravity to the prognosis. The same is true of organic disease of the heart.

A high range of temperature, 103° to 104° F., is unfavorable, as indicating pulmonary complications, although it has sometimes been observed that the febrile movement depended upon the bronchial disease alone, and abated as soon as the casts were expelled. Hæmoptysis, even when the amount of blood raised is considerable, is not usually of bad omen, because it ceases after the casts are thrown off. If the amount is very great, there is a probability of phthisis, although extreme hemorrhage has been noted in non-tubercular cases.

Failure to expel the casts must be regarded as somewhat unfavorable, since it prolongs the dyspnœa and cough which tend to exhaust

the patient. Fagge reported a case fatal through plugging of the windpipe by a loosened cast.

In general the acute form of this disease is more dangerous than the chronic, but this distinction is somewhat arbitrary, and it is difficult to say how many of the fatal cases regarded as acute might have assumed a more chronic form if they had survived the first attack. In the same way the paroxysms in the chronic form are often very acute in nature. In any given attack the danger to life is not very great except through complicating conditions already mentioned. But the prognosis as regards recurrence is very uncertain. It is impossible to say how often relapses will occur or how long the tendency to this disease will last. Days, weeks, or months mark the period of disability in different cases, and in a few instances, as before mentioned, there may be almost continuous suffering for several years, while in others there is a lifelong tendency to this distressing complaint. As in all pulmonary affections, the danger to life is greatest in young children and in old persons. Elbridge G. Cutler reports the following fatal case in a lady of sixty-five, with the autopsy:[1] The patient, whose general health was excellent, contracted a slight cold, which developed suddenly into an alarming attack that resulted fatally in a day or two. The post-mortem examination showed that "the pharynx and larynx were healthy. The tracheal mucous membrane in its lower half was injected and slightly thickened. A croupous membrane lay reflected on itself over the entrance of the two primary bronchi. When laid in place this membrane reached less than halfway up the trachea. Downward it extended into the minutest sections of the bronchi, in many of them forming an almost solid plug. The alveoli contained no solid matter, and, except in a few places where there was œdema or collapse, they contained air."

Extension of the membrane to the trachea is not common, and it must increase the danger of suffocation.

Treatment.—There is no mode of treatment which is of much avail in loosening the fibrinous exudate. Iodide of potassium has been tried in numerous cases for that purpose without noticeable benefit. An atmosphere charged with steam and the inhalation of atomized fluids may have some effect in softening the membrane. The chemical fact observed by Dixon more than a hundred years ago (1783), that the casts were soluble in alkalies, has led to the use of this class of remedies, especially lime water, internally and by vaporization. No brilliant results from this mode of treatment are recorded, nor has the use of mercurials been more successful. After the casts become detached expectorants or emetics are indicated. The subcutaneous injection of apomorphine, $\frac{1}{20}$ to $\frac{1}{6}$ grain, is the best form of emetic. The importance of sustaining the strength by general measures is obvious, also the necessity for such symptomatic treatment as will best relieve the cough, pain, and dyspnœa. Opiates are often invaluable. Change of climate is of doubtful expediency in most cases, except for an incidental general bronchitis.

[1] *Boston Medical and Surgical Journal*, vol. civ. p. 443, 1881.

BRONCHIECTASIS.

DEFINITION.—Dilatation of the bronchial tubes is not a separate disease, but is an anatomical lesion resulting from various affections of the bronchi and lungs.

It occurs under three general conditions:

I. As a congenital defect or anomaly. This form is always unilateral and diffuse, the whole bronchial tract of one lung being represented by a series of cavities opening into each other. It is very rare.

II. As an accompaniment of inflammation of the bronchi with weakening of their walls.

III. Through contraction of the lung tissue, as in interstitial pneumonia, or as the result of compression or collapse.

ETIOLOGY.—Bronchiectasis in the majority of cases occurs as the result of chronic inflammation of the bronchial walls, especially in the condition known as chronic catarrh. This results in atrophy of the muscular and elastic elements and a transformation of the wall into weak and yielding connective tissue. Hence it is unable to withstand the air pressure during cough, and gradually gives way. Accumulation of putrid and decomposing secretion tends to hasten this process, both by favoring degenerative changes and by mechanical pressure. The localized stenoses of the bronchi which so frequently occur in chronic catarrh also play an important part in the production of dilatation—if partial, by increasing the air pressure behind them during expiration; if total, by causing atelectasis and subsequent dilatation of neighboring bronchi. Foreign bodies in the air passages and obstruction from pressure, as by aneurysms, mediastinal tumors, or enlarged glands, have a similar action. Other frequent causes of bronchial dilatation are pulmonary tuberculosis, asthma, fibroid phthisis (interstitial pneumonitis), and the extensive adhesions which result from pleurisy. The two conditions last mentioned often coexist, and it seems clear that fixation of the pleural surfaces, with gradual contraction of the indurated lung, must cause dilatation of the bronchial tubes so far as their elasticity will permit. Bronchiectasis may also be caused by unresolved pneumonia.

PATHOLOGIAL ANATOMY.—Anatomically, the bronchiectases are divided into two main classes—the cylindrical and the saccular. The cylindrical are characterized by a uniform dilatation of the bronchi, occurring most commonly in the middle-sized tubes. The saccular are spherical or oval dilatations which are confined to definite areas of the bronchial tubes. The bronchus is sometimes obliterated, so that the dilatation forms a closed cavity. The cylindrical form is usually due to a long-continued bronchitis, and is frequently associated with emphysema. This is the form ordinarily met with in children, developing after measles or whooping cough. The saccular form, on the other hand, occurs more commonly as the result of atelectasis or induration and contraction of the surrounding lung tissues. This is often secondary to a previous pleurisy. Both forms, however, may exist side by side in the same lung.

The walls of the dilated bronchi show marked histological changes. The epithelium is more or less degenerated and changed in character,

in some cases taking the form of pavement epithelium. The basement membrane, however, remains intact for a long time. The muscularis is atrophied and the fibres are separated and replaced by fibrous tissue. The elastic fibres are also widely separated, stretched, and atrophied. The glandular tissues atrophy and disappear, and even the cartilages finally become involved. These changes are most marked in the saccular variety, some of the cavities apparently being lined with nothing but a thin membrane. In some cases, however, hypertrophic changes occur in the connective tissue and the mucosa shows papillary and band-like protrusions. Finally, the wall may break down and ulcerate. This occurrence is probably favored by the presence of decomposing secretions.

Sputum.—There is nothing especially distinctive about the sputum in the milder cases. In those where there are large saccular cavities, however, it is quite characteristic. It is very abundant, and is raised in large amounts after periods of retention. It is thin, purulent, grayish green in color, and, where decomposition has taken place, of a very foul odor. On standing it separates into three layers—the upper frothy, the middle thin and watery, the lower thick and granular. Microscopically, this is composed almost entirely of pus cells, with more or less numerous fatty epithelial cells and myelin particles, and many micro-organisms.

Symptoms.—While a slight uniform dilatation of the bronchial tubes, such as may occur in the ordinary course of chronic bronchitis, asthma, or emphysema, may give rise to no characteristic symptoms, it is different when there are larger bronchiectatic cavities either of the fusiform or sacculated variety. The accumulated bronchial secretion gives rise to an expulsive cough, usually most severe and paroxysmal in the morning, which is relieved only by the expectoration of large quantities of muco-purulent material, of varying character as described above, and sometimes so fetid as to be suggestive of gangrene or a foul-smelling empyema.

The degree of dyspnœa which may be present depends upon the amount of bronchial obstruction, the extent to which the pulmonary structure is impaired, and upon the complicating conditions of heart and lung which may develop as the disease advances.

Hæmoptysis, slight or severe, may occur, even in non-tubercular cases, from ulceration of the bronchial mucous membrane, but it is not frequent.

Fever, night-sweats, diarrhœa, and emaciation, caused by absorption of the retained and decomposing secretions, produce in some cases a cachectic appearance, as in phthisis. Further signs relating to the impeded pulmonary circulation in advanced cases are clubbed fingers and cyanosis. Dropsy if it occurs is usually the result of emphysema.

Physical Signs.—The percussion tone over large bronchiectases varies with the amount of fluid in the sac. It is dull when the cavity is full, but high-pitched and more or less tympanitic after an evacuating paroxysm of cough, especially if the site is superficial or near the apex. Deep-seated dilatations are hard to detect by percussion, and the sound elicited is often modified by emphysema or by induration of the adjacent lung substance.

Respiratory changes often noted are feeble expansion and diminished murmur, while, again, there may be prolonged expiration with a harsh broncho-vesicular tone. Large bronchiectatic cavities distended with air may give rise to cavernous or amphoric sounds which are very suggestive of tubercular excavation. In some cases there is a lack of vocal resonance and fremitus, and in others the vibration is increased.

In general the indications are those of bronchitis, with additional physical signs dependent upon the site, the extent, and the contents of the bronchial sacs, together with those signs which are caused by changes in the adjacent lung tissue through emphysema, consolidation, or shrinkage.

COMPLICATIONS AND SEQUELÆ.—The presence of bronchiectatic cavities, as already mentioned, may lead to hemorrhage through ulcerative changes in the bronchial wall. Abscess formation in the pulmonary substance may follow. Induration of the lung surrounding the bronchi is not uncommon; and pulmonary emphysema, with dilatation of the right side of the heart and consequent venous congestion of the liver, spleen, and kidneys, is a frequent complication at a late stage in the disease. In prolonged cases amyloid changes in these organs may occur. In rare instances the adjacent lung tissue becomes gangrenous, and metastatic abscesses of the brain have been recorded.

Pulmonary Osteo-arthropathy.—The peculiar hypertrophy of the ends of the long bones, especially the terminal phalanges, which in recent years has been termed chronic hypertrophic pulmonary osteo-arthropathy, may result from the long continuance of residual decomposition in bronchiectatic cavities. The bulbous enlargement of fingers and toes presents an appearance not unlike that in acromegaly, but the nose and chin are not involved, as in that disease, and the bones themselves are the seat of the hypertrophy, as is shown by the *x*-rays. The cause of this condition is doubtful. It has been attributed to the absorption of toxins from suppurative processes in the lungs, pleura, or bronchi, and has been observed chiefly in tuberculous excavations, empyema, and chronic bronchitis with bronchiectasis.

DIAGNOSIS.—The diagnosis of bronchiectasis depends upon the history of the case, the physical signs, and the symptoms, as already enumerated, and the exclusion of tubercular cavities, pulmonary gangrene, and empyema.

In children a chronic cough with signs of emphysema, following an attack of whooping cough or pneumonia, is suggestive of bronchial dilatation. In adults, especially in elderly persons, a history of long-continued bronchitis, emphysema, or interstitial pneumonitis, with the characteristic expectoration and physical signs above mentioned, renders a diagnosis of bronchiectasis highly probable.

This condition can be distinguished from pulmonary phthisis with cavity formation by the absence of tubercle bacilli from the sputa, and also by the fact that tubercular cavities are most frequently at the apex, while dilated bronchial sacs more commonly have their seat in the lower portions of the lung. Moreover, the tubercular excavation is preceded, often for a long period, by signs of consolidation, as well as by the rational signs of tuberculosis. Excessive fetor of breath and expectoration is sometimes as noticeable in bronchiectasis as in gangrene of the

lung, but this latter affection is usually secondary to tuberculosis, pneumonia, or pulmonary embolism, and is characterized by rapid systemic prostration and alarming constitutional symptoms which indicate the gravity of the disease. In gangrene, as in pulmonary abscess, the presence of elastic fibres in the sputa serves to indicate that the lung substance is invaded.

A localized empyema discharging through the bronchi may give rise to the same profuse, paroxysmal, fetid expectoration which is observed in bronchiectasis. The physical signs may not suffice for a clear diagnosis between these two conditions, but careful study of the history and symptoms of the case, with the bacteriological examination of the sputa, will often lead to a correct decision. Such empyemata are usually secondary to pneumonia, and pneumococci are often found in the expectoration. Exploratory puncture may be necessary to determine the diagnosis.

In uncomplicated bronchiectasis the temperature has a negative value for diagnostic purposes, since it is seldom much elevated, and the constitutional symptoms, except in far-advanced cases, are comparatively slight.

The presence of bronchiectatic cavities which develop behind strictures or narrowings of the bronchial tubes may be inferred from the characteristic symptoms and signs and the previous existence of a tumor, aneurysm, or specific disease which by pressure or cicatricial contraction would cause the obstruction.

In pulmonary actinomycosis (a disease which has rarely been diagnosticated during life) there is a pervading fœtor of the sputa when the bronchial passages are involved, as in bronchiectasis, but the ray fungi are found in the secretions.

Prognosis.—Bronchial dilatation may continue for many years with remissions and exacerbations of the symptoms, but it is practically incurable. Its course may be mild or severe, according to the age and endurance of the patient and the presence or absence of complicating pulmonary conditions. In some instances a considerable degree of bronchial dilatation is not incompatible with a fair amount of exercise or work. Cases with pulmonary consolidation, especially of the interstitial form, with atelectasis or extensive pleural adhesions, progress most rapidly, while the acute dilatation which occurs in children after whooping cough or measles affords a better prospect for recovery, owing to the greater elasticity of the tubes and the absence of chronic changes in the bronchial mucous membrane.

Treatment.—The objects of treatment are to maintain the general health of the patient, to facilitate expectoration, and to correct the fetid odor. Those remedies which are found useful in chronic bronchitis are valuable in a limited way only in this condition. Inhalations containing creasote, tar, terebene, turpentine, eucalyptus, or menthol are more or less effectual as antiseptic and deodorizing agents.

Bronchiectatic cavities or sacs which result from occlusion or narrowing of the entering bronchi are usually dependent upon causes which are little amenable to treatment, such as aneurysms, tumors, or syphilitic contractions. In some cases of this description iodide of potassium affords relief, in doses of 5 to 30 grains *ter die*.

Surgical interference is justifiable when the physical signs point to the presence of a large accessible bronchial sac with fetid contents which is walled off by pleural adhesions. Under these circumstances treatment by incision and drainage is applicable for the prevention of septic absorption from the retained secretions, as in cases of localized empyema, provided the general state of the patient is such that the operation in itself is less dangerous than the continuance of the existing conditions. A few successful cases have been recorded in which this mode of treatment has been undertaken. In a larger number the fatal result has been hastened, and the prognosis is by no means so favorable as in the surgical treatment of pulmonary abscess from acute causes. The occurrence of cerebral abscess has been noted in some cases thus operated upon, but also in other cases of long-standing fetid bronchiectasis, as the result of pyæmic infection.

ASTHMA; HAY FEVER.

By A. LAWRENCE MASON, M. D.

ASTHMA.

DEFINITION.—Asthma is a neurotic affection characterized by spasm of the bronchial muscles, and associated, in many cases, with hyperæmia and turgescence of the bronchial mucous membrane and an exudate of mucin. The essential predisposing cause is an unstable condition of the respiratory centres, the nature of which is at present unknown. The paroxysms may be due to direct irritation of the bronchial mucous membrane, or may be excited reflexly by irritation of any of the branches of the pneumogastric nerve and its communications, or even of other nerves. They may also originate in stimuli transmitted from the higher cerebral centres, and, probably, from irritations of the respiratory centres themselves by certain substances in the circulating blood.

THEORIES REGARDING ASTHMA.—There are at present two main theories as to the nature of the obstruction in the air passages. The first theory, chiefly associated with the names of Hyde Salter and Biermer,[1] is that the bronchial muscles, especially in the smaller bronchi, contract spasmodically at various points and remain in a state of spasm for a longer or shorter time. Weber's[2] theory, however, supposes that there is a sudden swelling of the bronchial mucous membrane through acute dilatation of its bloodvessels, resulting from vasomotor paralysis.

Sir Andrew Clark[3] went farther, and said that "the paroxysms begin by a more or less diffused hyperæmic swelling of the bronchial mucous membrane, and are continued by the development at various parts thereon of circumscribed congestive swellings, which come and go with greater or less rapidity, resembling the skin in urticaria." Biermer, while considering bronchial spasm as the chief element, nevertheless recognizes the etiological importance of hyperæmia of the mucous membrane. The anatomy of the parts helps us greatly in deciding between these theories. Reisseissen and Kölliker demonstrated the presence of muscular fibres in both large and small bronchi, and soon after Williams[4] showed that irritation of the lung caused contraction of these fibres. Subsequently, Longet and Volkmann produced a constriction of the bronchi by galvanizing the pneumo-gastric. Roy[5] and Graham Brown have recently shown that the vagi contain

[1] Biermer, *Volkmann's Sammlung. klin. Vorträge*, Leipzig, 1875.
[2] Weber, *Versamml. deutsch. Naturforscher u. Aerzte*, Leipzig, 1872, p. 159.
[3] Clark, *Internat. Journal Med. Sciences*, Jan., 1886.
[4] Williams, *Path. and Diag. of Diseases of the Chest*, London, 1840.
[5] Roy and Brown, *Transactions of the Physiolog. Society*, vol. ii., Appendix, p. 21.

fibres which both constrict and expand the bronchi. It seems evident from these experiments that there are cerebro-spinal centres which control the contraction and relaxation of the bronchial muscles.

Auld [1] has recently shown that in all the membranous bronchi the diameter of the wall is one-seventh that of the lumen. In the larger bronchi, also, the diameter of that part of the wall comprising the mucosa and submucosa is also about one seventh of the lumen at the corresponding point. The advocates of Weber's theory claim that the swelling of the mucous membrane is sufficient to account for all the symptoms, and in support of this they advance the frequent association of coryza and asthma and the phenomena of vaso-motor congestion in the nose, as well as the alternation of hay fever and asthma. Störck [2] by laryngoscopic examination saw the mucous membrane of the trachea grow red with the onset of the paroxysm and resume its normal appearance with the subsidence of the attack. The secretion of mucus which soon occurs is thus held to be sufficient to account for a part of the obstruction. There is no doubt that a swelling of the bronchial mucous membrane, with a greater or less exudation of mucus, takes place in every case of asthma. There is likewise little doubt that these phenomena are the result, and not the cause, of the paroxysm. In the first place, it is evident from the anatomical structure of the bronchial walls that a congestion sufficient to cause marked stenosis is impossible. Moreover, no such stenosis is found either in œdema or acute inflammation of the bronchi. In the second place, the nasal mucosa with its cavernous structure is in no way comparable with the bronchial mucosa; and, lastly, many attacks of asthma are terminated by the expectoration of only a very small amount of mucus.

From the results of the experiments of Roy and his predecessors there can be no doubt that a spasmodic contraction of the bronchial muscles is possible. Moreover, we have an analogous condition in the spasmodic contractions of the smooth muscle fibres of the intestine. The sudden changes in physical signs are also readily explained by changes in the localization of the spasms.

Wintrich and his followers, notably Bamberger, claim that the paroxysms are due to tonic spasm of the diaphragm alone or in connection with the other muscles of respiration. They hold that in tonic bronchial spasm the chest should be collapsed and the diaphragm elevated. Biermer has answered this assertion by showing that the spasmodic constriction in the bronchi acts as a valve which allows the entrance of air during inspiration, but prevents its escape during expiration. If the expiratory pressure were exerted on the contents of the alveoli alone, it would easily overcome the constriction, but it also compresses the bronchioles and tends to close them more tightly. It thus happens that the diaphragm is forced downward by the distention of the lungs, but it does not remain fixed in one position, as would be the case if it were in a state of spastic contraction. Lebert,[3] however, believes that there are secondary spasmodic contractions of the diaphragm and other respiratory muscles of the neck and chest.

[1] Auld, *Path. of Bronchial Affections and Pneumonia*, London, 1891.
[2] Störck, *Mittheil. über Asthma bronchiale*, Stuttgart, 1875.
[3] Lebert, *Klinik der Brustkrankheiten*, Bd. i. S. 438.

Leyden[1] concluded that the Charcot-Leyden crystals cause the bronchial muscle cramp by mechanical, and perhaps also by chemical, irritation of the bronchial mucous membrane. Then Curschmann[2] called attention to the spirals which bear his name, and to the fact that the crystals were not always present. He opposed the supposition of Leyden that the crystals caused the paroxysms by direct irritation, on the ground that they are most abundant in the deepest layers of the spirals and very rare on the surface, and considered the spirals to be the cause of the attacks through an exudative process in the bronchi, which he called "bronchiolitis exudativa." Other observers soon found the crystals in other diseases as well. Von Jaksch, Vierordt, and Pel then showed that the spirals were also found in other conditions, and that the sputum might be loaded with them without any attack resulting. Schmidt[3] has recently shown that they can no longer be regarded as more than an accompanying symptom of various inflammatory processes of the respiratory tract. (See Figs. 17, 18, pp. 170, 171.)

Lazarus[4] and Leyden[5] still think that the crystals have a direct causative action in the production of the paroxysms of asthma, and Müller is inclined to the opinion that they have something to do with them. The presence of the crystals without an attack is explained by assuming that a certain predisposition through the neurasthenic peculiarity of the patient is necessary to an asthmatic seizure. Müller called attention to the preponderance of eosinophile cells in asthmatic sputum, and with Fink[6] showed an excess of eosinophile cells in the blood of asthmatics—an observation which was confirmed by V. Noorden.[7] Gollasch and Seifert consider Charcot's crystals to be crystalline products of the eosinophile cells. Müller, however, thinks that the crystals are not formed from the eosinophile cells, but that they are the products of some other substance which has a positive chemotaxic action on the eosinophile cells, and that the excess of these cells in the blood is due to the same chemotaxic action. Other observers regard the presence of the crystals and cells in the sputum as no proof of a special form of disease.

Experiments by Gerlach tend to show that no specific disease is necessary for the formation of spirals, but only a certain degree of tenacity in the sputum, as he has produced these spirals by taking an end of sputum in forceps and twisting it. Hence he concludes that their formation is the result of axial torsion, and that they are formed in the lungs by the to-and-fro movement of air, this air stream acting on the free ends of sputum so as to twist them. Ruge thinks that the central fibres begin to be formed in the bronchioles and are twisted a little there, becoming more so and taking on their outer coat in the larger bronchi.

It should be stated that the theory of bronchial spasm as the explanation of asthma has not met with universal acceptance in recent years. Thus, Berkart,[8] in his valuable treatise based on large experience,

[1] Leyden, *Virchow's Archiv*, 1872, Bd. liv. S. 324.
[2] Curschmann, *Deutsches Archiv f. klin. Med.*, 1883, Bd. xxxii. S. 1.
[3] Schmidt, *Zeitschrift für klin. Med.*, 1892, Bd. xx. S. 476.
[4] Lazarus, *Berlin. klin. Wochenschr.*, 1891, S. 899.
[5] Leyden, *Deutsch. med. Wochenschr.*, 1891, S. 1086.
[6] Fink, *Diss. Bonn*, 1890.
[7] V. Noorden, *Zeitschrift f. klin. Med.*, 1892, xx. 98.
[8] Berkart, *On Bronchial Asthma*, London, 1889.

regards the dyspnœal paroxysms as a symptom merely of a peculiar inflammatory affection of the bronchi accompanied by a croupous exudate, which by purely mechanical obstruction produces stenosis of the bronchi. The bronchial plug coming from below upward is thought to act as an expiratory valve, allowing the access, but preventing the egress, of air, and producing what may be termed a "bronchial tenesmus." On this theory Berkart considers the subjective dyspnœa and the forcible respiratory movements as chiefly the consequence of deficient ventilation of the lungs, although he admits that irritation of the sensory fibres of the vagus may play some part in the production of these symptoms. This author further regards it as highly probable that a streptococcus observed by him is the exciting cause of the progressive inflammation peculiar to bronchial asthma, in a manner analogous to that which is seen in erysipelas of the skin.

In reviewing these various hypotheses which have been advanced to explain the nature of asthma it seems probable that bronchial spasm, due to inflammation and obstruction in the bronchi themselves or induced by reflex irritation transmitted from some more distant part, impedes the respiration, and, through deficient expiratory action, allows the accumulation of carbonic acid in the respiratory centres. This again excites the pneumogastric function, the lungs become distended, and the action of the diaphragm is thereby impaired; these various causes producing a state of suffocation which is relieved only by relaxation of the complex spasmodic element—a result which often follows the expectoration of a relatively small quantity of tenacious mucus.

Etiology.—The etiology of asthma in the individual is to be sought primarily in the above-mentioned personal idiosyncrasy depending upon a respiratory neurosis. Further than this, in many cases there is a lesion, morbid condition, or functional disturbance in some part of the respiratory tract or in some more distant organ (stomach, intestine, uterus), which by direct or reflex irritation establishes the tendency to asthmatic spasm.

But it is not always possible to refer the beginning of asthma to any special cause, constitutional or local. There is a difference between the origin of the disease and the origin of the paroxysms, the former being now under consideration. Reference will be made later to the many causes which excite spasms in asthmatic subjects.

Idiopathic Asthma.

Etiology.—In a certain number of cases, marked by the regularity and severity with which the paroxysms reappear, asthma can be traced to no antecedent disease or present condition in which the symptoms might find their exciting cause. Heredity plays an important part in the etiology of primary asthma, which occurs usually in persons of an unstable nervous organization. Thus, worry, fright, exposure, or fatigue may bring out the asthmatic tendency in those whose immediate ancestors have also been subject to asthma or to some other neurotic ailment. The trouble may come from a grandparent, the morbid tendency never having developed in the parent, and in some

cases asthma shows itself in children born long after the active manifestations of the disease in the parent have ceased.

Different opinions have been held as to the frequency with which asthma really occurs as an hereditary disease. Some writers are skeptical in the matter. But the best authorities regard the hereditary proclivity as a strong factor, Hyde Salter, for instance, having observed it in two fifths of a series of cases. Berkart found that asthma was a family inheritance in 30 cases, 16 per cent. of his series, the patients of both generations having been under his observation—a fact of importance, since much of the evidence on this point is from hearsay. This author attaches great weight to the rachitic conformation of the thorax in the parents and children, as showing that hereditary asthma and the anæmia which so often accompanies it are manifestations of the rachitic diathesis.

Asthma is common in families with a strumous or phthisical history, and in other families certain individuals may suffer from asthma and epilepsy in turn, or the asthmatic paroxysms may alternate periodically with neuralgic attacks. This, with the fact that asthma has been observed in the children and grandchildren of persons who have developed epilepsy late in life, would appear to indicate as the cause a common neurosis of cerebral origin.

Age.—The influence of heredity and of certain other causes which are more or less potent in the development of the asthmatic neurosis is often manifest at an early age. Salter met with 7 cases in infants under a year old, some of them truly congenital. During childhood the predisposition to asthma is called into activity by the acute exanthemata and the various respiratory diseases incident to early life, to which later reference will be made. At the time of the first or second dentition and at about the age of puberty asthma may declare itself. Many patients, however, who pass through their childhood and early youth with no sign of this disease, become typical asthmatics during the second, third, or fourth decade. After the age of forty relatively few persons acquire the disease without a concurrence of bronchitis, emphysema, or some other affection of the air passages to which the asthma is evidently secondary. Exceptionally, individuals in advanced years, sixty to seventy, develop asthma without apparent cause of a local or reflex nature.

Sex.—It is commonly stated that many more males have asthma than females—according to some authors in the proportion of two to one. This does not accord with what would be expected in a disease of neurotic origin. But the difference between the sexes in this respect is not very marked, and in the experience of some observers female asthmatics have been more numerous, while others refer the disproportion in favor of the male sex at certain ages to accidental causes (Berkart; Powell).

In infancy the disease is rare, and males and females may be alike affected. During the first decade, when asthma makes its appearance in more than a third of the cases, more boys than girls suffer from it, as they do from bronchitis and many other acute diseases. But during the period of sexual development the more sensitive female organization shows a greater proclivity to asthma, especially in its purely neurotic

form, and occasionally the menopause calls forth the asthmatic tendency. So asthma may first appear during pregnancy. In Berkart's experience 5 patients had stated that their asthma commenced during gestation. Toward term they had been seized with severe coryza, cough, and then paroxysmal dyspnœa. All, however, had marked hereditary predisposition to asthma and had been subject to severe sneezing fits. The asthma of later life which is secondary to pulmonary affections is much more common in men.

Pulmonary Affections.—Besides these general relations of age, sex, and heredity, pulmonary affections have an important bearing in determining the asthmatic habit either through bronchial irritation from atmospheric or other causes, or as a sequence of pneumonia and bronchopneumonia. It cannot be said, however, that the tendency to this complaint is produced, fostered, or perpetuated by any general atmospheric conditions to which all subjects are susceptible. Neither a damp, foggy air, nor the constant respiration of a dusty, smoky, or impure atmosphere, nor the sudden alternation of heat and cold, is sufficient to originate the disease in any considerable number of cases, and it is probable that most asthmatics become so without much regard to their surroundings. The statement of Salter that in the largest number of cases the worse the air for the general health the better it is for asthma, is applicable only to the effect of city air on the frequency of the paroxysms in a certain number of individuals. The absence of ozone is held to have some bearing upon this point.

The effect of pulmonary diseases in producing the first manifestations of asthma is seen in numerous cases in early life when pneumonia, bronchitis, or those diseases in which bronchitis is a conspicuous element, such as whooping cough and measles, mark the starting point for a lifelong asthma.

It is not clear whether these diseases are to be regarded as merely the determining causes of the asthmatic tendency in persons of unstable respiratory power, the subjects of a "bulbar neurosis," or whether the morbid changes in the lung and bronchial membrane are sufficient to create this tendency and to leave behind a permanent liability to asthmatic spasm, although the restoration of the lung function is otherwise complete. It is probable that in many cases this causative relation is of a secondary exciting nature only. The onset of asthma in children after bronchitis, whooping cough, or measles seems to be often attributable to the pressure of enlarged bronchial glands. An analogous explanation serves to account for the asthma of strumous subjects and for those cases of spasmodic dyspnœa which are associated with aneurysmal, mediastinal, and cervical tumors where there is direct pneumogastric irritation.

It is not known that any structural changes result from pneumonia by which a subsequent asthma might be provoked, unless pleural adhesions can be regarded as a probable cause. Moreover, the interesting fact is observed that in confirmed asthmatics, during an attack of pneumonia, the spasmodic paroxysms remain in abeyance, as do those of whooping cough when pneumonia supervenes, until after the resolution of the pulmonary consolidation, when they recur as before.

Throat and Nose Affections.—Adenoid growths in the pharynx may

cause asthma, and in recent years much stress has been laid upon the nasal origin of this disease. Voltolini's cure of a severe case by the removal of a nasal polypus led to subsequent investigation, which has brought about a clearer recognition of this reflex source of asthma. Bosworth goes so far as to assert that asthma, in a large proportion of cases, is attributable to obstruction in the nose from polypi, swollen turbinates, and other inflammatory conditions, which, through the agency of the sensory distribution of the fifth nerve, by reflex sympathy cause bronchial spasm. He fortifies this assertion by the reports of a large number of cases cured or greatly relieved by local treatment applied to the nose, and these views have been widely adopted and corroborated. The relation between paroxysmal sneezing, coryza, hay fever, and asthma has also become more obvious. But, admitting that nasal obstruction or inflammation is, in a certain number of cases, a causative factor in the production of asthma, it is probable that these conditions are not the sole cause, but that they are often engrafted upon the deeper-seated neurosis, which, according to other observers, remains active in many cases after the nasal affection has been cured. Moreover, there is great practical difficulty in determining the antecedent conditions under which asthma has developed, since most patients come under observation after the disease has fully declared itself, and the supposed relations of cause and effect may be due to coincidence.

Gastric, Cardiac, and Renal Affections.—Gastric and intestinal disturbances of a functional character, such as dyspepsia or worms, appear to hold some causative relation to asthma, either through reflex pneumogastric irritability or through the absorption of deleterious substances by the blood (ptomaïnes).

True asthma is seldom associated primarily with cardiac disease, but the dyspnœa which attends all forms of heart affections often bears a resemblance to asthma in its spasmodic quality. The same is true of the so-called renal asthma, a term which is often applied to dyspnœa of uræmic or toxæmic origin, similar to that which occurs in diabetes, probably from ptomaïne absorption. It is chiefly with the gouty type of kidney, nephritis of the interstitial variety, that paroxysmal dyspnœa is observed, and the occasional association of asthmatic attacks with lead-poisoning is also noticeable. Workers in lead are prone to gout, and these mutual relations are all suggestive of toxæmia from faulty elimination by the kidneys.

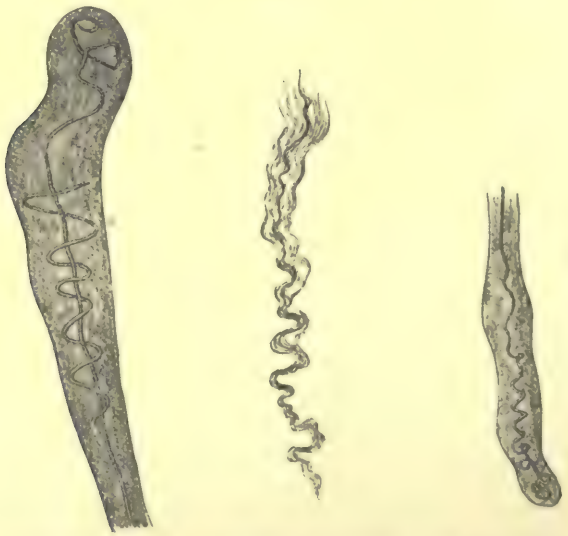
Gout and Skin Affections.—The onset of asthma is apparently due in a few subjects to the gouty diathesis, with or without bronchitis. Cases are recorded in which asthmatic paroxysms, previously very severe and frequent, permanently disappeared after the occurrence of acute arthritic gout. In other instances gout and asthma have alternated. A similar coincidence or alternation of asthma with certain forms of skin disease has been too often observed to be fortuitous. Trousseau, himself a sufferer from hereditary asthma, in his graphic description notes the frequency with which such diathetic transformations take place, rheumatism, gout, hemorrhoids, gravel, and various skin affections being in certain individuals replaced by asthma and replacing it in turn. The disappearance of skin eruptions to which patients have long been subject on the appearance of asthma marks an interesting feature in the

etiology of this complaint which has provoked considerable discussion. The evidence of the best observers, both clinical and dermatological,[1] goes to show that this transference of symptoms is more than a coincidence; also that asthma under these circumstances is not a reflex phenomenon due to skin irritation, but that there is an underlying dyscrasia, either in the nervous system or in the blood, upon which both the respiratory and the skin affections depend. Eczematous and herpetic eruptions, urticaria, psoriasis, and acne are the skin diseases which most frequently show an association with asthma, but it is noticeable that these are very common skin affections, and that their connection with asthma is on the whole rare.

Occupation and Mode of Life.—While asthma is certainly more common among professional men who lead a sedentary life, and in general among the better classes with sensitive nervous organizations than among outdoor laborers, still it is often met with among the poor both in its hereditary and secondary forms. High livers of full habit are prone to asthma, although the typical appearance of the chronic sufferer is thin, high-shouldered, round-backed, and nervous.

Nervous Origin.—From these general considerations it is evident that the etiology of asthma is complex. The belief that in all cases

FIG. 17.



Curschmann's spirals (magnified 275 diameters; after Eichhorst).

it is dependent upon a neurosis is not based upon definite knowledge as to the special nerve centres involved in its production, whether in the brain, the medulla, or in the pneumogastric and sympathetic systems. From the great diversity of external impressions which excite the paroxysms, often almost instantaneously by reflex wave from some distant point, and from the alternation in some cases with other neuroses

[1] Bulkley, *British Med. Journal*, 1885, vol. ii. p. 954.

(epilepsy, hemicrania), a functional instability in the higher nerve centres seems probable in many instances.

PATHOLOGICAL ANATOMY.—The lungs present no change especially characteristic of asthma. In long-standing cases we find emphysema and evidences of chronic catarrh, both of which are due to secondary affections and not to the primary disease. As noted above, Störck has observed hyperæmia of the larynx, trachea, and bronchi during an attack, but there is no proof that this leads to permanent tissue changes. No changes have been found in the nervous system sufficient to account for the disease.

Sputum.—The sputum is quite distinctive. Early in the attack it consists of small, translucent, ball-like bodies floating in a little thin mucus, which are known as the "perles" of Laennec. Later it becomes muco-purulent. When these balls are unfolded they are seen to represent moulds of the smaller tubes. Microscopically, they have a spiral, twisted form (Fig. 17), and are composed of a clear substance with cells entangled in it. This substance is mucin. Others show in addition a clear filament in the centre which is also composed of mucin. These spirals, discovered by Curschmann, were supposed by him to be characteristic of the disease, but, as previously stated, they have since been found in many other conditions. (See page 165.)

The Charcot-Leyden crystals, which have already been referred to, are pointed, colorless, octahedral crystals (Fig. 18). They are identical with the crystals to be seen in post-mortem blood and with those found in the semen and in the blood in leucæmia. They are the phosphate of æthylenimin or diæthylendiamin.[1]

FIG. 18.

Charcot-Leyden crystals in the sputum of an asthmatic (× 300; Charcot).

SYMPTOMS.—The symptomatology of asthma relates to the characteristic peculiarities of the paroxysms, their exciting causes, and to the conditions which persist during the interval. This comprehends both the acute and chronic, as well as the idiopathic and catarrhal forms, since chronic changes take place sooner or later in the great majority of cases, and catarrhal symptoms of more or less severity are rarely absent, even in cases of purely nervous type at the outset. The catarrhal element predominates over the spasmodic in some instances, chiefly in children.

The history of the paroxysm is that of a prolonged struggle for breath, with a constant and terrifying sense of impending suffocation. All of the usual and accessory muscles of respiration are taxed to the utmost. The breathing is labored, the inspiration being short, strong, and spasmodic, the expirations prolonged and feeble from the ineffectual effort to get rid of the residual air. The diaphragm may be fixed, and sometimes its action is reversed, rising with inspiration and falling with expiration, while the falling in of the soft parts with each respiratory act shows how little air finds entrance in spite of the intense dyspnœal exertion.

Many postures are assumed to gain relief, the thorax usually being

[1] V. Jaksch, *Clinical Diagnosis*, 1893, p. 110.

thrown forward and often fixed by grasping some support. Ordinarily the respirations are not increased numerically, and they may be less frequent than normal. Speech is difficult and broken; the alæ nasi are distended; the face is pale, sometimes cyanotic; and the beads of perspiration which stand out upon the forehead, with the protruding eyes and anxious countenance, make an alarming picture, more suggestive of suffering and danger than is seen in any other functional disturbance. Dry, harassing cough is a prominent feature of some attacks at an early period, attended later by expectoration, and finally by the expulsion of small plugs of mucin, sometimes bloodstreaked; but hæmoptysis is rare. The bodily temperature may be normal, elevated (101° to 103° F.), or it may be even several degrees below the normal. The pulse is usually small and rapid. Urine of a low specific gravity is passed freely, sometimes involuntarily from the strong abdominal contraction. After a time, varying from a few minutes to several hours, the symptoms abate either gradually or, it may be, suddenly, after the bronchi are relieved of the obstructing exudate. Occasionally fibrinous casts are expelled, and the occurrence of the spirals of Curschmann and the Charcot-Leyden crystals has already been mentioned. (See page 171.)

During the attack there is often much nervous agitation, even fright, in those who are as yet unfamiliar with the symptoms, but delirium is not a feature of this complaint. The mode of access varies. In some cases there are prodromata in the form of headache, drowsiness, or gastric disturbances; in others there is more or less preliminary coryza, wheezing, and cough, while often the paroxysm begins with no conscious warning whatever, usually in the latter part of the night. The patient then awakes with a sense of suffocation, which impels him to walk the room or seek an open window for more air. A peculiar reflex symptom sometimes observed in the beginning of an attack or during the partial dyspnœal seizures to which asthmatics are subject is itching under the chin. This same sensation may be felt between the shoulders or over the sternum. It appears with the first feeling of pulmonary constriction, and subsides with the development of the paroxysm.

In some cases a single paroxysm exhausts the tendency for the time being, like a fit of epilepsy or migraine; in others there is a marked periodicity dependent upon the return of an exciting cause—the menstrual flow, for instance—but, as a rule, there is no great regularity either in the intervals between the attacks or in their duration. Often fresh paroxysms occur night after night for a week or two, or even for a much longer time, and when the habit is once established there is little variation in the hour at which the seizures take place. Trousseau says that he was awakened by a sense of oppression, and always heard the clock strike three, while his mother was seized between six and eight o'clock in the morning. Diurnal attacks are, however, exceptional, and are usually attributable to faulty digestion or to organic disease of the heart or lungs.

As the paroxysm passes off the patient falls into the sleep of exhaustion, but if there is no recurrence appetite and strength, in early cases, are little impaired, and during the intervals life goes on as usual. The tendency in individuals is to a repetition in each attack of the features which have characterized preceding attacks, and in that way

asthmatics can sometimes tell about how often their attacks will come, the time of onset, and how long they will last. There are, however, many other cases of purely idiopathic asthma in which the paroxysms recur with the utmost irregularity after intervals of days, months, or years.

The prodromata to which allusion has been made relate chiefly to cases in which gastric or respiratory disturbance gives warning of the impending explosion, possibly in time to avert the crisis by remedial measures. But no adequate impression of the capricious nature of this disease can be conveyed without reference to some of the exciting causes which precipitate the paroxysms with little or no warning. These are the whole class of irritants to the respiratory mechanism, either through direct bronchial inhalation or by way of the olfactories, and certain obscure reflex agencies which cause paroxysms through their impression upon the nervous system.

EXCITING CAUSES.—Although asthmatic subjects individually may show great susceptibility to atmospheric states, the subtle differences which cause the attacks or hold them in abeyance are largely arbitrary. Damp, foul, smoky air may not provoke them; neither may the clear air of the mountains prevent them. Many sufferers find relief in large cities, even in circumscribed portions of cities. They have asthma in certain houses and not in others. A place which gives immunity to one person may be bad for another, and wherever the first experience is unfortunate, there the paroxysms will usually recur time after time. Even in favorable localities it often happens that sooner or later unknown causes arouse the tendency and a change becomes necessary, while places that have been the scene of previous suffering may subsequently afford comparative or entire comfort.

The respiration of irritant gases or vapors will in some persons immediately bring on an attack, while in others the exciting cause is found in the dust from oats, rice, or linseed meal. Similar to this is the hay asthma which aggravates some cases of hay fever in summer.

Curious idiosyncrasies are observed in the production of asthmatic seizures by powdered ipecacuanha, as related by Cullen, Watson, Trousseau, and others, and by certain odors of which some persons are intolerant. Trousseau states that the smell of a bunch of violets in a room always gave him asthma. Flint says of himself that the peculiar emanations from feather beds or pillows brought on attacks of coryza, bronchitis, and asthma, although he never experienced the least degree of asthma from any other cause. Many cases are related by Hyde Salter, Beard, and Flint in which the emanations from horses, dogs, cats, rabbits, and other animals provoked asthmatic paroxysms. In consequence of this peculiarity, which may be hereditary, some persons cannot go to horse-shows, dog-shows, or menageries. Salter mentions a case in which the application of cold to the instep always caused an attack. Nervous shock, mental emotion, or a fit of laughter may be sufficient to start a paroxysm, and these same conditions of nervous disturbance may give rise to erythema or urticaria—a fact which has some bearing upon the causation of the so-called "eruptive asthmas."

COMPLICATIONS AND SEQUELÆ.—In asthma of the idiopathic, purely spasmodic type, especially in young and otherwise healthy subjects, there is generally complete restoration to health in the intervals

between the attacks. The paroxysms pass off, and such patients engage in their usual pursuits with unimpaired lung function. Tennis, rowing, and mountain-climbing do not disturb their respiratory equilibrium. But even in this class of cases, sooner or later, the frequent recurrence of asthmatic attacks tends to produce permanent emphysema and some degree of bronchial catarrh, while wheezing persists during the intervals.

Cases of more decidedly catarrhal type present these conditions in greater intensity, often with bronchiectasis and dilatation of the ventricles, so that eventually there is constant dyspnœa with periodical exacerbations, besides the secondary venous congestions which the disturbed circulation entails. Asthma does not predispose to phthisis, and the cases are rare in which sufferers from this disease become tubercular.

Diagnosis.—The diagnosis of asthma is based upon the history of the case, the paroxysmal nature of the seizures, the character of the dyspnœa, and the physical signs.

Most cases are established and unmistakable when they are first seen, but in children the history is not always clear, the attacks are often atypical, and from the predominance of catarrhal symptoms the spasmodic element may be overlooked. A history of asthma in the parents or grandparents, the discovery of any reflex source of irritation in the upper air passages or alimentary canal, and the favorable course of the attacks will usually serve to indicate their origin, although for a time they may pass for spasmodic croup or bronchitis of the finer tubes.

In youth and adult life the paroxysmal seizure, its suddenness and rapid development, and the excessive dyspnœa, with the physical signs which accompany it, are in most cases sufficiently distinctive. The absence of laryngeal cough and obstruction usually gives evidence that the dyspnœa is not inspiratory, except so far as the already distended condition of the lungs prevents the entrance of more air, while the pulmonary dilatation, the impaired action of the diaphragm, and the long, distressing expiratory effort are in marked contrast to other respiratory affections.

Physical Signs.—The contour of the chest and shoulders observed in asthmatics, the appearance and the positions assumed by the patient, have already been mentioned. The characteristic physical signs in asthma distinguish it from pneumonia, pleurisy, phthisis, and extensive primary bronchitis. The signs relate both to auscultation and percussion. The respiratory murmur is heard feebly throughout the chest, accompanied by loud râles, sibilant and sonorous, and at a later period by moist râles, which attend the secretion and expulsion of mucus. The expiratory sound is greatly prolonged, low-pitched, and of vesicular quality, in distinction to the bronchial breathing of pneumonia. The percussion tone is everywhere intensified on account of the pulmonary dilatation, and extends over the cardiac space and to a lower limit than normal. Vocal resonance and fremitus may be unchanged or diminished. The heart sounds are rapid and feeble, and in cases where dilatation of the right ventricle exists the impulse is noticeable at the epigastrium. Cardiac impulse in this region is due also to the depression of heart and diaphragm by the distended lungs. If it is suspected that the dyspnœa may be caused by obstruction at the larynx, the use of the

mirror will determine this point. It seldom happens that the dyspnœa produced by the pressure of aneurysms or intra-thoracic tumors is mistaken for asthma.

PROGNOSIS.—The immediate prognosis in any given paroxysm of asthma is entirely favorable. It is hard to find record of a considerable number of fatal cases. In spite of the alarming aspect which patients present, their lividity, intense suffering, and exhaustion, the impression prevails that asthma is never directly fatal. Berkart, however, states that death occurred in a paroxysm in 5 cases of his series, or 3 per cent., and that information was wanting as to the exact mode of death in a number of other cases. These fatalities were in persons of middle age or beyond who had suffered from asthma since early life, and who had developed secondary changes in the lungs, heart, or kidneys.

The younger the subject, as a rule, the better is the prospect for recovery. Those developmental conditions which in children promote bronchitis, chorea, and other nervous disorders also tend to excite asthma, and this tendency may diminish during adolescence. It is not safe, however, to assure parents that their children will "grow out of it." Very often they do not, and the habit acquired in childhood is a permanent one. Favorable circumstances are the absence of hereditary proclivity, the presence of a remediable local cause, sound organs, and mild attacks. During youth and adult life the chances for recovery diminish, and after middle age cure is rare.

Emphysema, chronic bronchitis, and cardiac dilatation preclude all chance of permanent improvement, and these conditions in many instances, combined with frequent long asthmatic attacks, surely tend to shorten life. While some persons with this complaint live to a great age, this is exceptional. Asthma is not conducive to longevity. That is often dependent upon heredity and unusual resistance to the secondary organic changes and constitutional impairment which this disease entails.

In general, the frequency and severity of the seizures determine the degree and rapidity of the secondary changes, but to this there are many exceptions, in which violent attacks occur at longer or shorter intervals, year after year, with little effect upon the general health and no pulmonary or cardiac symptoms during the intervals. An intercurrent attack of extensive acute bronchitis or of fibrinous pneumonia is very dangerous.

TREATMENT.—The relief and abridgment of the paroxysm is the most urgent indication which presents itself in the treatment of asthma, but no less important is the management of the patient during the intervals between the attacks with regard to prophylaxis, so far as that may be practicable, and the care of complicating conditions.

In the paroxysm the sense of impending suffocation demands more air. Free ventilation partially supplies this want, and may be supplemented by oxygen inhalations if, after trial, they appear to do any good. In the mean time, it is well to use those remedies which are at hand and which the patient has found alleviating in previous attacks, unless they are positively harmful, as, for instance, are morphine and chloral if frequently administered. Strong coffee is worth a trial. Most patients

have their own favorite remedies in which they place faith, and these are often the best for them, at least for the time being. The great number of "asthma cures," however, and the fact that many patients are disposed to try them all, affords the best proof of their uncertainty and empirical action.

Rational indications point to the removal of any local cause for irritation which may exist, either direct or reflex, and to the relief of spasm by narcotic or sedative drugs. Indigestion after late suppers not infrequently provokes asthma toward morning, and in some cases unloading of the gastro-enteric tract by an emetic and enema shortens the paroxysm. The most convenient way to produce emesis is by the subcutaneous injection of apomorphine, $\frac{1}{10}$ to $\frac{1}{5}$ grain, which will empty the stomach in ten or fifteen minutes. It also relaxes spasm and provokes expectoration. Great debility and a feeble heart are contraindications to the use of this drug. Apart from those cases in which there is evident digestive disturbance, the production of nausea by repeated drachm doses of antimonial or ipecac wine or of the wine of lobelia is effectual in stopping the spasm in a certain number of cases. When the source of irritation is in the naso-pharynx a spray of cocaine solution, 2 to 4 per cent., should be tried, and transient relief at least may be attained.

In most cases, for the control of the paroxysm, some of the more strictly antispasmodic drugs or combinations will be found desirable. Many of these when in the form of fumes act largely as irritant expectorants.

No remedy is so potent for the relief of the dyspnœa in purely spasmodic cases or in those associated with a dry, harassing cough as morphine sulphate or valerianate given subcutaneously. Its action is so prompt and soothing that patients soon become dependent upon it, and obvious objections arise which are sufficient to condemn the use of this drug in all chronic ailments. The effect of the remedy may be worse than the disease, and many asthmatics are so fully aware of this danger that they are unwilling to seek relief in this way. Morphine does not relieve all cases, but sometimes appears to aggravate the dyspnœa, probably by checking the bronchial secretions. Atropine, $\frac{1}{100}$ grain, is often combined with morphine, $\frac{1}{8}$ to $\frac{1}{4}$ grain, or given separately, and when the paroxysms are infrequent and moderate doses suffice, with none in the intervals between the attacks, these drugs may be effectual in controlling to some degree the asthmatic habit.

Ether and chloroform to the extent of primary anæsthesia are sometimes indicated, either at the beginning of a severe paroxysm, when these remedies are known to be successful in breaking up the spasm, or later in an exhausting attack which resists other methods. Chloroform is dangerous for subjects with weak hearts.

Nitrite of amyl by inhalation, 3 to 5 drops on a handkerchief, from its action upon the vaso-motor system relaxes spasm and flushes the face. This in the very pallid type of asthmatics often gives transient relief, as does nitro-glycerin in repeated doses of $\frac{1}{100}$ grain in tablets, and occasionally the nitrite of sodium in 5-grain doses. Hyoscyamus is often combined with these drugs. Iodide of ethyl, to be inhaled during the paroxysm, was strongly recommended by Germain Sée. It is most easily given, like amyl nitrite, by breaking 5-drop "pearls" in a

handkerchief and inhaling from time to time. By this means the intensity of the attack is interrupted, but the action is not lasting.

Chloral hydrate, or a combination of chloral with bromide of potassium, in doses of 10 to 30 grains each, is a powerful agent for checking asthmatic dyspnœa, but in long paroxysms the doses usually have to be renewed to an amount which increases the subsequent depression, and may even be dangerous in subjects with feeble circulation.

The action of stramonium, like that of belladonna, in doses of 10 to 20 minims of the tincture, is often very satisfactory in allaying spasm, but with both these drugs caution is necessary to avoid toxic effects. Stramonium is used, at one time or another, by almost all asthmatics for the benefit derived from its fumes. The dried leaves are burned in the room, which must be tightly closed, or they are smoked in a pipe or in the form of cigarettes. Stramonium, belladonna, and hyoscyamus, with a small proportion of opium, enter into the composition of most asthma cigarettes, of which those of Espic, Joy, and others have long been in high repute. They often fail, and their virtue appears to be due to increased cough and expectoration set up by the inhaled fumes, as well as to the sedative action of the drugs absorbed. The same is true of the various "asthma powders." Tobacco-smokers are, as a rule, less benefited by inhaling the asthma remedies than others, and vice versâ in non-smokers the fumes of a cigar may be alleviating. Powell states that a powder containing 4 drachms each of powdered nitre and anise seed and 5 grains of tobacco has proved useful at the Brompton Hospital. A teaspoonful of this powder is burned upon a plate and inhaled through a large inverted funnel. In the same manner the fumes of saltpetre paper, prepared by soaking blotting paper in a strong solution of saltpetre, afford an easy mode of relief for some asthmatics.

The application of ice bags over the pneumogastrics in the neck or to the upper spinal region is sometimes successful in allaying the paroxysm, and finally all the various idiosyncrasies of asthmatic patients regarding the influence of light, darkness, temperature, etc. must be carefully considered.

As before mentioned, the endless number of drugs and prescriptions which have been tried, with more or less benefit, for the relief of the asthmatic paroxysm shows how difficult it is to establish any rules of practice which will not very often fail, and for this reason quack remedies and nostrums find in the chronic sufferers from this disease their most constant patrons.

Most of these secret remedies contain iodide of potassium in large quantities, a drug which is often resorted to in doses of a drachm or two daily during the intervals between the attacks, and sometimes with considerable benefit. Germain Sée held it in high esteem.

Treatment during the Intervals.—When the paroxysm is over, if exhaustion is great, there may be a continuous state of dyspnœa with fever which is favorably influenced by complete rest, systematic feeding, tonics, and stimulants. The subcutaneous injection of full doses of strychnine, $\frac{1}{60}$ to $\frac{1}{30}$ grain, is to be recommended. Arsenic has met with favor as a preventative of frequent paroxysms in the form of Fowler's solution, 3 to 5 drops after meals. So iron, quinine, and sulphur for

their restorative effect upon the blood, and bromide of potassium for its influence upon the nervous system, are deserving of trial.

The tendency to recurrence of the attacks is to some extent dependent upon the degree of bronchitis or of emphysema which may coexist, and success in prophylaxis is proportionate to the relief afforded to these conditions. It is in this direction that the iodide of potassium and the syrup of hydriodic acid exert their chief influence, although these preparations sometimes prove useful when given in an entirely empirical manner.

Emphysema, when not attended by advanced cardiac changes, is more or less remediable by respiratory gymnastics and the use of some form of pneumatic cabinet or compressed air chamber, as recommended by Salter. Good results have been reached in many instances with Waldenburg's apparatus or its modifications, from the alternate inspiration of condensed air and expiration into a rarefied atmosphere.

Careful examination of the nose and throat should be made in all cases, in view of the many recorded cures after the removal of adenoids, polypi, and swollen turbinates. In fact, this must be regarded as the chief advance of recent years in the hitherto unsatisfactory treatment of asthma, although the limitations to the success of these operative measures are now recognized.

Every precaution should be taken by the asthmatic subject to maintain the best bodily condition possible. Bathing and friction for the skin, alkaline mineral waters with occasional saline laxatives, a nutritious but moderate diet, and outdoor exercise are to be enjoined. If, in spite of all efforts, there is a tendency to increased frequency of the paroxysms, a change of climate must be advised. Many patients know the climates which are best for themselves and seek them at regular intervals. The high altitudes of this country and of Europe, such as Colorado and the Engadine, bring relief to many cases not yet emphysematous, the relaxing mid-ocean resorts to many others, and in general it may be said that a dry, inland air is often good for those who suffer near the sea, and that the seashore moisture sometimes relieves patients from the interior. The benefit derived is frequently proportionate to the improvement in the bronchial catarrh which has become persistent, or to the gouty and rheumatic conditions in those patients who seek the various spas for a periodical cure.

HAY FEVER.

Definition.—Hay fever is a neurosis, often hereditary, characterized by extreme susceptibility of the air passages at certain seasons to the action of various irritants. Occasionally it is attended by asthmatic paroxysms.

Etiology.—Besides the neurotic element essential to hay fever, the predisposition, a necessary etiological factor is the summer season, and finally there must be an immediate excitant, such as the pollen of grasses, flowers, or ragweed. Some specialists claim that an abnormal state of the nasal membrane is always present as the chief causative factor. In many cases hypertrophic rhinitis is found, in others polypi or deviation

of the septum; but these conditions are present in many persons who do not have hay fever, and it is probable that such changes are often the result of continuous irritation rather than the cause of the disease in question. It seems reasonable, however, to suppose that pathological conditions of the nose favor periodical recurrence of the attacks.

Since the first description of this disease by John Bostock[1] in 1819, numerous valuable works in this country and in Europe have satisfactorily determined its etiological and seasonal relations, geographical distribution, course, and treatment. The theory that hay fever is exclusively due to the pollen of flowering grasses and plants was elaborated experimentally with great care by Blackley,[2] in opposition to the wider view of Phœbus[3] and Pirrie,[4] who had suggested that other external agencies, such as heat, strong light, and ozone, also acted as exciting causes. The belief that the central nervous system was a more important factor in the production of hay fever than had been supposed was also advanced by Pirrie, and met with support in the valuable treatise of Morrill Wyman,[5] whose contributions to this subject began at about the middle of this century. Wyman regards the sudden onset, tumultuous course, and transitory character of hay fever, together with its peculiarities in time of commencement and duration, and the fact that little alleviation follows any medical treatment except that addressed to the nervous system, as indications that the cause may act first upon that system, perhaps principally upon the great sympathetic.

The investigations of Beard[6] further established the belief, which is now generally accepted, that "hay fever is primarily and essentially a neurosis."

It is noticeable, however, that many of the later contributions to the literature of this subject in America, especially those by Bosworth, J. Mackenzie, and Sajous, tend to minimize the importance of the neurotic element in hay fever as compared with the more obvious nasal lesions which are said by these writers to be its chief cause. It is held that stenosis of the nasal passages so far impairs the respiratory function of the nose that the spongy, erectile tissues over the turbinates, the so-called "turbinate corpora cavernosa,"[7] become engorged, and a state of "rhinitis vaso-motoria" ensues under the influence of the various external irritants which are concerned in the production of hay fever. Later, reference will be made to the results of treatment based upon this mechanical theory.

Passing to the consideration of more general etiological conditions, it is evident that the subjects of hay fever often acquire it by inheritance. The facts which sustain this view are, as Beard states, "of a most overwhelming character." Wyman, himself a sufferer, records numerous cases in his own family through four generations. It makes

[1] Bostock, "Catarrhus Æstivus," *Med.-Chirug. Trans.*, vols. x. and xiv.

[2] *Experimental Researches on the Causes and Nature of Catarrhus Æstivus*, by Charles H. Blackley, M. R. C. S., London, 1873.

[3] *Typischer Frühsommer-Katarrh, oder des sogenannte Heufieber, Heuasthma*, Giessen, 1862.

[4] *Hay Asthma and the Affection termed Hay Fever*, London, 1867.

[5] *Autumnal Catarrh (Hay Fever)*, New York, 1876.

[6] *Hay Fever or Summer Catarrh*, by George M. Beard, 1876.

[7] Bigelow, *Boston Med. and Surg. Journal*, 1875.

its appearance in many cases in early childhood and in individuals of a nervous diathesis. Many of the subjects of hay fever have also other nervous disorders of a functional kind, such as migraine, neuralgia, and insomnia, less frequently chorea or melancholia.

Age and Sex.—Hay fever may first show itself at any time between childhood and middle age. In most cases the tendency declares itself before the age of twenty-five, and instances are comparatively rare in which the first appearance is delayed until after the age of forty. Blackley says that "it never comes on so late in life," but in this country both Beard and Wyman mention a number of cases as beginning after fifty. It is not easy to estimate with accuracy the relative proportion of males and females who are affected by this complaint, since men are much more exposed to its various exciting causes, such as dust, smoke, pollen, and heat, but, so far as statistics go, they tend to show a much smaller proportion of females—about 1 female to 3 males. There is some ground, however, for thinking that females are more susceptible than males to the early form, "rose cold," which prevails in May and June.

Occupation.—There is no doubt that merchants, professional men, and persons of sedentary habits, brain-workers, supply most of the victims to this malady, which is rare among farmers or laboring men either in the city or country, although an outdoor life in the rural districts implies a much greater exposure to the usual exciting causes. Most of the cases occur among the better class of society, who can afford to run away during the season for the attacks, but it is not so uncommon now as it formerly was to meet with cases in the hospital out-patient departments both here and in England.

Nationality.—The Anglo-Saxon race shows an especial proclivity to hay fever, and it is more common in this country than in England. Cases are rare among the foreign population in our cities, whether German, Swede, French, or Italian, and among the negroes and Indians it is practically unknown.

Season.—The hay fever season in America lasts from May until November, its limitations being chiefly dependent upon the stage of vegetation of the different plants which act as exciting causes. In England it may last from May until September, but the usual time is in May and June, when the pollen of grasses and flowers is most abundant. In this country the early form, or "rose cold," usually begins in the first week in June and lasts for two or three weeks, coinciding with the flowering period of early blooming plants and the new-mown hay.

Some persons, however, suffer all through the middle of the summer from other kinds of vegetation. The late form, the "autumnal catarrh," so fully described by Wyman, is that which most commonly prevails in America, and its season corresponds with the depressing heat of the "dog-days" and the flowering time of certain late-blooming plants. There is little doubt that the pollen of Indian corn, golden rod, and especially of Roman wormwood or ragweed (Ambrosia artemisiæfolia), is an excitant of those catarrhal attacks which begin about the 20th of August and last until October or in some cases until the cold weather. It is thus apparent that, while the season for individuals depends upon their

own idiosyncrasies in regard to different irritants, there are certain pervading influences which regulate the time and duration of the disease in its several forms.

Geographical Distribution.—Hay fever prevails most extensively in the New England, Middle, and Western States this side of the Mississippi River. It is less common in Maryland, Virginia, the border States, and the Far West, and is rarely seen in the extreme South or on the Pacific Slope. The Rocky Mountain region is exempt. The zone between the thirty-fifth and forty-fifth parallels of latitude practically includes the hay fever district, although cases occur north and south of these limits. In this extensive section there are many localities which from their elevation, proximity to the ocean, or from the absence of certain forms of vegetation confer immunity upon hay fever sufferers. These places will be mentioned with the curative regions in connection with the treatment of hay fever.

Exciting Causes.—While the pollen of certain grasses and plants already mentioned is doubtless a frequent cause of hay fever, most subjects are not sensitive to the emanations from hay itself. Dust of all kinds, bad air, heat, sunlight, and gaslight cause great annoyance to many. Railway smoke, brimstone matches, flowers, and fruits, especially peaches, are intolerable to others. In fact, the nasal mucous membrane is so hypersensitive that the least pungency of odor or chill of temperature may suffice to aggravate the symptoms. The influence of the imagination may be enough in the susceptible to bring on an attack. Phœbus relates the case of a patient who had sneezing and other characteristic symptoms "while looking at a beautiful picture of a hay field." The presence of an artificial rose in the room caused similar phenomena in a patient of Mackenzie, and other cases equally curious, as showing the strength of mental influence, are recorded. Indeed, it is probable that the recurrence of this disease year after year on the same day may be attributed in part to the effect of fixed anticipation.

No distinctive bacteria have yet been discovered.

PATHOLOGICAL ANATOMY.—The pathological changes which take place are thought to be due to vaso-motor paralysis which follows the action of the various irritants upon the nasal membrane. Extreme vascular dilatation with much swelling ensues, so that the nostrils are occluded and there is a profuse serous transudation. Other nasal changes sometimes found, such as deviation of the septum or chronic rhinitis, cannot be regarded as characteristic of hay fever.

SYMPTOMS.—The symptoms in most cases declare themselves at about the same time every year, even on the same day and hour, it is said by some sufferers from "autumnal catarrh." The first indication is often a sensation of itching or tickling in the nose, mouth, throat, eyes, and ears, which is soon followed by a feeling of discomfort in the region of the frontal sinuses. Frequent transient paroxysms of lachrymation, sneezing, and nasal obstruction occur in a day or two, provoked by slight causes to which the now hyperæsthetic membrane is susceptible, and in the milder cases passing off at this early stage without great discomfort. Soon, however, the catarrhal inflammation of eyes, nose, and throat becomes intense and painful; a profuse, watery discharge escapes

from the nostrils; a troublesome conjunctivitis is established; and the patient becomes so generally miserable that physical or mental effort is next to impossible.

There is great variation in the severity of the symptoms in different cases. Some subjects can, with a good deal of effort and extreme care in prophylaxis, pursue their customary avocations for a week or two, or even during the whole period of susceptibility; others have high fever, headache, and prostration to a degree which disables them entirely and confines them to the house.

When the attack is fully developed the nostrils become almost occluded by the great swelling of the nasal mucosa; the flow is excessive, and is relieved but temporarily by the paroxysms of sneezing which occur at intervals. Difficulties of breathing and of swallowing add to the discomfort, so that there is little rest day or night. Insomnia is common. It is often attended by a high degree of nervousness and a sense of suffocation which is out of proportion to the gravity of the conditions present.

Cough is not a constant feature of hay fever, but in a considerable proportion of cases it comes on in the second week and lasts through the attack. In some cases it is spasmodic, and so incessant at night that sleep is impossible, and the straining of the diaphragm and intercostals causes much soreness and pain. It does not usually give rise to the signs of extreme bronchitis, and expectoration is absent or scanty until a late stage. Cough may continue, however, long after the other symptoms of hay fever have ceased.

Asthma.—Asthma is a late symptom which in many cases comes on after the cough has lasted for some time and the most acute catarrhal symptoms have abated. It may appear, however, at the height of the disease, and is rather more common in "autumnal catarrh" than in the earlier forms of hay fever. The asthmatic period, as a rule, begins at about the fourth week of the disease, and the seizures do not vary from those in ordinary asthma. In some cases it is periodic, occurring at the same hour night after night, and under these circumstances it may be the most trying feature of the disease. The paroxysms appear to be more directly associated with antecedent bronchial irritation than with the nasal symptoms, but in some cases they occur as nasal reflex phenomena independently of cough. More commonly there is persistent cough in the intervals between the paroxysms of dyspnœa. Beard states that four fifths of the hay fever sufferers have cough or asthma, or both, but it should also be said that these symptoms may not recur with equal severity every year. In some seasons they may be entirely absent.

Complications and Sequelæ.—There is little tendency to permanent ill effects from hay fever, except the thickening of the nasal mucous membrane which results from prolonged irritation. The senses of taste and smell may be impaired during the attack and for a long time afterward. A general irritability and nervousness may also be more or less persistent. In a certain number of cases, usually in elderly persons who have suffered from hay asthma for many years, the heart becomes weak and intermittent during the attacks. This condition may pass away with returning health and strength or it may

result in permanent cardiac dilatation. Pneumonia is mentioned by Wyman as having occurred in 3 cases during attacks of hay fever. In 1 instance the catarrh ceased for two weeks, to return after the pneumonia disappeared, when asthma also came on for the first time. A similar amelioration of symptoms has been observed when the natural course of hay fever has been interrupted by severe accidents or other acute illnesses. Urticarial and herpetiform eruptions with intense pruritus give great annoyance to some patients.

Diagnosis.—Hay fever will seldom be mistaken for an ordinary attack of coryza or bronchitis, except in children and in the earliest attacks of those persons who have not yet recognized its seasonal periodicity. The sudden occurrence of the symptoms, previously detailed, in their regular sequence and intensity during the time of year when hay fever prevails, with the absence of the usual history and physical signs of acute bronchitis, will determine the diagnosis between these two affections. Confirmatory evidence may be obtained from the history of previous attacks at the same season, the observation of the effects of certain irritants, and the cessation of symptoms on removal from their influence or on the approach of cold weather.

Prognosis.—The prognosis as regards life is invariably good. Hay fever sufferers often live to a great age, and this disease is in itself no bar to life insurance. But the chances of getting permanently rid of the attacks is very small. When the tendency is once established, it recurs annually with great regularity, unless the patient removes himself from the exciting influences before the season arrives. To this rule there are few exceptions. The time of the attacks may change from an earlier to a later period; they may be less severe for a number of years in succession, and in some few instances they grow milder with advancing years. These facts must be taken into consideration in estimating the value of local treatment adopted during the intervals.

Treatment.—Treatment by change of climate alone promises complete relief, but for those who cannot go away much may be accomplished by local and constitutional measures. At the outset frequent preliminary washing of the eyes with a 2 per cent. boric-acid solution or some other non-irritating collyrium is advisable. The catarrhal inflammation of the nose is temporarily relieved in most cases by an ointment containing from 2 to 4 per cent. of cocaine. The inhalation of iodine, eucalyptol, creasote, carbolic acid, and other pungent remedies is sometimes effectual in checking paroxysms. For frequent nasal inhalation, however, it is better to use the vapor from a jug of boiling water to which a drachm of tincture of iodine or compound tincture of benzoin has been added.

Touching the sensitive areas repeatedly with the galvano-cautery or chromic acid is practised at this stage with benefit in some cases. The intra-nasal injections of weak solutions of quinine or hydrogen peroxide may also be tried. In severe cases, when there is complete occlusion from contact of the turgid nasal surfaces, these local measures are often ineffectual and avoidance of the exciting causes must be attempted. Satisfactory results sometimes follow a residence in the city, because vegetation is less abundant and journeys to and fro are discontinued. The patient should remain in a cool, quiet, closed room as much as possible, restraining the inevitable desire to blow the nose, as this always

aggravates the trouble. When travelling is necessary, the insertion of absorbent cotton, wet with a weak solution of cocaine, into the nostrils affords some protection against the irritation of smoke and dust.

Constitutional treatment by tonics and electricity for two or three weeks before the attack tends to make the patient more resistant. The internal remedies which have been found most useful for this purpose are quinine and arsenic in full doses. Iron and cod-liver oil are indicated for anæmic and feeble subjects. During the attack some persons find benefit from whiskey and other alcoholic stimulants. Morphine, atropine, chloral, the bromides, cannabis indica, and other sedatives may be required for the control of cough and asthma or to procure rest and relief from pain. This is often an essential part of the treatment. (See Treatment of Bronchitis and Asthma, pp. 133 and 175.)

Fortunately, it happens that many persons can prevent their attacks by going away, especially those who suffer in August and September. A sea voyage is curative, and a trip to Europe may be undertaken with almost complete confidence. A yacht cruise along the eastern coast is comfortable except when the wind is off shore. The New England seashore resorts and the islands, such as Fire Island, Block Island, Nantucket, and the Isles of Shoals, give more or less protection to many of the milder cases in the summer (June cold) and to some cases of "autumnal catarrh." But land breezes often cause relapses, and for the majority a more northerly and cooler resort is better. Some patients escape at Mt. Desert, and can remain there in comfort until October, while others cannot. Safety may be found farther to the eastward, at Campobello and St. Andrews, New Brunswick.

Many prefer to seek the mountains, and in one or another of these elevated regions immunity is obtained in most cases if they go before the attack begins and stay long enough. Elevation, a cool temperature, and absence of the vegetable irritants which excite the symptoms are the chief requirements for any given case, but the quality of the air and obscure atmospheric variations have some bearing upon the degree of relief, since slight changes of locality in the same region may produce very marked differences in the feelings of patients.

As a general thing, the northern mountains are the best for most patients. The Adirondacks and the White Mountains are preferable to the mountains of North Carolina and Virginia or the Alleghanies and the Catskills, although all of these regions are frequented. A sojourn of several weeks in the great forests of Northern Maine at an elevation of a thousand feet or more above the sea is usually protective. Moosehead Lake is the centre of this region. The advantages to be derived from "camping out" for those whose tastes enable them to enjoy that mode of life are an additional recommendation to the forest resorts. But in some cases all these places fail and comfort can only be found still farther north. Hay fever is almost unknown in Canada, and it is a very rare thing that complete immunity is not obtained in the Maritime Provinces and the province of Quebec. All parts of New Brunswick and Nova Scotia afford a delightful summer climate. The cities of Halifax and Quebec are pleasant resorts, now much visited by sufferers from hay fever, and on the lower St. Lawrence are many places which fulfil all the climatic conditions necessary for the comfort of the

victims to this ailment. But luxuries are not abundant and quarters are often primitive throughout this region—a serious matter for persons in search of health. Among the most desirable places are Murray Bay, Cacouna, Rivière du Loup, and the Madawaska country, now accessible by rail from the north and south.

How far hay fever is curable by local treatment of the nasal cavities during the intervals is a question which is still *sub judice*. The association of nasal stenosis, caused by hypertrophic rhinitis, polypi, and deviation of the septum, with hay fever and asthma appears to be more common in America than in England. Some rhinologists claim better results from local treatment here than are attained elsewhere. Conservative opinions are based upon the facts that this association is often accidental or secondary, that in many early cases it does not exist, and that after removal of the nasal obstruction the symptoms of hay fever, in a considerable number of cases at least, relapse after two or three years. Other cases disappear or fall into different hands, so that it is extremely doubtful whether a complete and permanent cure of the symptoms and the tendency is as frequent as has been supposed. Bad results are sometimes observed from adhesion of the nasal surfaces after too severe or hasty cauterization.

Nevertheless, it is obvious that special treatment should always be adopted for the correction of chronic nasal stenosis when it exists, and palliation of the hay fever attacks at least may usually be expected. For further researches in this direction the reader is referred to the writings of Bosworth, Sajous, Morell Mackenzie, Greville MacDonald, and other eminent laryngologists.

HÆMOPTYSIS.

By ELBRIDGE G. CUTLER, M. D.

Definition.—True hæmoptysis (from *αἷμα*, blood, and *πτύσις*, a spitting, spitting of blood) is indicative of a hemorrhage into the respiratory apparatus somewhere between the larynx and the pulmonary alveoli. The source of the hemorrhage, its cause, its amount, and its duration vary very greatly. It is, therefore, a symptom rather than a disease. The blood may either appear with a slight expiratory effort in the mouth and be spit out, or it may gush forth from the nose and mouth in considerable quantity, or, lastly, it may be raised by acts of coughing. Pseudo-hæmoptysis, or spurious hæmoptysis, is where blood is spit which originally came from some point outside the respiratory tract, as the nose, mouth, stomach, or œsophagus, and, having reached the pharynx, has gravitated into the air passages.

The blood in true hæmoptysis may come from the larynx, trachea, bronchi, or lung. Hemorrhages from the larynx or trachea are not frequent. They are observed in certain catarrhal conditions and ulcerative processes, in aneurysms of the aorta, and in aneurysms of the pulmonary, subclavian, or carotid artery which have perforated these parts of the air passages. Hæmoptysis of the latter class is profuse and uncontrollable, and is rapidly fatal through suffocation or the amount of blood lost. For a more extended description the reader is referred to that part of this work which treats of Aneurysm (p. 568). In most cases the hemorrhage is from the bronchi or from the lungs, and we shall confine our attention to this part of the respiratory apparatus in what follows. Of these two sources of hemorrhage, that from the bronchi is most frequent, but many times it is wellnigh impossible to determine with certainty whether the blood comes from the bronchi or the lungs.

Etiology.—The most frequent occasion of bronchial hemorrhage is tuberculosis, the cause assigned being partly relaxation of the bronchial mucous membrane and liability to laceration of its bloodvessels, partly fatty changes in the bloodvessels, partly growth of tubercle in the wall of the vessels with subsequent disintegration. It is not extremely rare for hemorrhage to occur in young healthy persons when no other physical signs can be detected and nothing further follows. Such cases are mentioned by numerous writers. In a very large number of cases of hæmoptysis, however, tubercle bacilli are found at an early stage, and the case goes on from bad to worse.

Other causes of bronchial hemorrhage are inflammatory and ulcerative processes on the bronchial mucous membrane. A severe bronchial catarrh and also fibrinous bronchitis may give rise to hæmoptysis. It also occurs in diseases of the heart as a result of passive congestion,

most frequently in lesions of the mitral valve, aortic stenosis, and tricuspid insufficiency, though it does occur in disease of the other valves and in disease of the heart muscle. Hæmoptysis is, moreover, found in bronchiectasis, and especially in putrid bronchitis.

A further source of hæmoptysis is severe mechanical, thermic, or chemical irritation of the bronchial mucous membrane. Under mechanical irritants may be grouped severe coughing, loud and continued speaking, singing, shouting, loud sneezing, carrying heavy weights on the back or lifting them, forced mountain-climbing, dancing, riding, gymnastic over-exertion. The inspiration of very hot or very cold air may cause hæmoptysis; also the breathing in of irritating gases. If foreign bodies have got into the bronchi, they may either cause hæmoptysis at once by direct injury to the mucous membrane, or hemorrhage follows an ulceration consequent on the loosening and expulsion of the foreign body.

Perforations of aneurysms of the aorta or pulmonary artery into a bronchus may give rise to hemorrhage, as before indicated.

Traumatic bronchial hemorrhage is rarely seen because of the protected position of the bronchi.

Many cases of bronchial hemorrhage depend on general nutritive disturbances, as hæmophilia, scorbutus, purpura hæmorrhagica, cholæmia. Others occur in acute infective diseases, as measles, scarlet fever, smallpox, when they are of the hemorrhagic variety. Bronchial hemorrhage is found sometimes in a certain form of intermittent fever which has been described as febris perniciosa hæmoptoica. In such cases the hemorrhage occurs daily at a particular time, and disappears either spontaneously or after the use of quinine, in the former cases assuming other more pronounced malarial symptoms.

Vicarious bronchial hemorrhages have been described where the hæmoptysis occurred in place of bleeding from some other organ; the only one which bears rigid investigation is hæmoptysis instead of menstruation, which has been very frequently observed.

Hæmoptysis often follows exposure to rarefied air, as in mountain-climbing and balloon ascents.

Hemorrhage from the Lungs.—The most frequent cause of pulmonary hemorrhage, as of bronchial, is pulmonary consumption. The hemorrhage here is sometimes capillary and sometimes arterial, in the latter case being a late symptom and usually indicating the presence of a cavity, It is not rare to find in the wall of cavities dilated or aneurysmal arteries, the rupture of which has given rise to profuse hemorrhage. A thrombus may form at the point of rupture, which may thus check the hemorrhage temporarily or permanently. In the former case a loosening up or displacement of the thrombus may follow which allows the bleeding to recur afresh.

Besides pulmonary consumption, gangrene or abscess of the lungs may give rise to hæmoptysis when bloodvessels which are still permeable are reached by the ulceration. The hemorrhage in gangrene of the lung is apt to be very profuse, and, according to Fräntzel, moreover, may be the first sign of the disease.

Hæmoptysis is not a rare occurrence in tumors, as cancer and sarcoma of the lung, and in parasites of the same, especially echinococcus.

It is also endemic in parts of China and Japan, due to the presence of the Distoma Ringeri in the bronchial tubes, and it is said to have been caused by the presence of filaria.

The sputum may be mixed with blood in acute inflammatory disease of the lungs, as in the first stage of fibrinous pneumonia, and sometimes also in catarrhal pneumonia.

Injuries to the lungs commonly though not necessarily cause the form of hemorrhage called pulmonary apoplexy; at the same time there may or may not be accompanying injury to the bones and soft parts of the thorax.

Aneurysms which have enlarged at the expense of the lung may rupture and give rise to pulmonary laceration and enormous hemorrhage.

Very frequently pulmonary hemorrhage occurs in heart disease as hemorrhagic infarct. The anatomical changes are not always the same in these cases. In rare cases it is a rupture of pulmonary capillaries; more often it depends on embolism or thrombosis. Emboli most frequently come from portions of cardiac thrombi which have their origin in the right auricular appendage or cardiac wall near the right apex. Mitral and tricuspid lesions are the ones oftenest accompanied by changes in the right side of the heart, so that hemorrhagic infarct chiefly follows trouble with these valves. It may be encountered, however, in other valvular lesions and in changes of the right side of the heart following pulmonary emphysema. Hemorrhagic infarct and hæmoptysis may also depend on thrombosis of the peripheral veins, the embolus reaching the right side of the heart through the superior or inferior vena cava.

In some cases the hæmoptysis seems to be of nervous origin, the result of vaso-motor influences perhaps. Certain experimenters have caused pulmonary hemorrhage in animals by injuring definite portions of the cerebrum. Other observers have recorded hæmoptysis as occurring in cerebral hemorrhage, in mental diseases, chorea, epilepsy, hypochondria, and in disease of the brain and spinal cord. Hysterical hæmoptysis has been described by some of the old classic writers, and it has been observed by modern authors as well.

Experience shows that hæmoptysis is more common in men than in women, and that it occurs oftenest from the fifteenth to the thirty-fifth year. It is very rare in children and in aged persons.

PATHOLOGICAL ANATOMY.—The anatomical changes vary with the nature of the hemorrhage.

In bronchial hemorrhage the bronchi contain more or less blood, which is sometimes fluid and sometimes clotted, and may look either fresh or black or brown. The bronchial mucous membrane frequently appears to be swollen and friable. It may be deeply injected, or anæmic and pale if the hemorrhage has been abundant. As the hemorrhage is almost always a capillary one, bleeding points are not found, as a rule. Some blood is usually aspirated into the alveoli, so that it is not always possible to say in any given case that all of it has come from the bronchi and not partly from the lungs as well. Red blood corpuscles may also pass from the alveoli into the pulmonary connective tissue—an absorptive process which may be quite extensive and rapid.

Of the different forms of hemorrhage from the lungs the pathological condition in pulmonary apoplexy is the simplest. In this form a

greater or smaller portion of lung is composed of blood and lacerated lung tissue.

In hemorrhage from tuberculous cavities a dilated vessel may often be found in the wall of the cavity with its point of perforation either open or plugged by a thrombus, and the cavity itself and connecting bronchi filled with fluid blood or clots.

In hemorrhage from the lungs of capillary origin, as a rule it is not possible to designate the precise spot whence the blood came. A number of bright red, dark red, or, later, brown red nodules are found in the lungs, which are dense and solid to the touch, but are often surrounded by emphysematous lung. On section these nodules have a slightly granular surface, and microscopically the alveoli and infundibulum walls are found to contain red blood corpuscles. The epithelium cells of the alveoli are not unfrequently slightly swollen, have granular contents, and are more or less desquamated. After a time, if absorption has not occurred, the corpuscles undergo destructive changes. They shrivel, become granular, and blood pigment in the shape of needles or plates becomes deposited in the alveolar wall or infundibular septa.

Pulmonary hemorrhage in cardiac cases seldom comes from rupture of bloodvessels; it generally depends on embolic processes. The shape of such a hemorrhagic infarct is usually that of a wedge, with its apex toward the root of the lung and its base toward the pulmonary surface, whence its name of "wedge-shaped infarct." Such infarcts are usually small and are located near the outer surface of the lung, though they may also occur deep near the root. When on the surface the pleura is inflamed. The embolic infarct feels hard to the touch, contains no air, on section is dark blue or brown red, somewhat granular, and on scraping it with a knife a bloody mass is obtained which the microscope shows to consist of blood corpuscles. An infarct may undergo any one of several secondary changes. It may dry up, absorb, and leave behind a brown or black pigmented connective tissue, or desiccation and caseation or softening, formation of abscess, or gangrene may result. If the pleura is involved in the destructive process, pneumo-thorax may follow. Several infarcts are often found in the lung, frequently in different stages of development. They are chiefly seen in the lower lobe of the right lung, a favorite place being between the scapular line and the spinal column, according to some authorities. Less often they occur on the left side or in the middle or upper lobes. Eichhorst thinks the power of the blood current and the weight of the embolus are responsible for its ultimate position in the right lower lobe. If from previous embolism the current to the right lower lobe is weakened, or if the right pulmonary artery is diminished in size by contraction of the lung or pressure of a pleural effusion, or the current is lessened from any other cause, embolism takes place on the left side. It is not always possible to find an embolus in the vessel on careful search. On the other hand, it is not necessarily the case that every embolus causes hemorrhagic infarct.

If the embolism depends on thrombus of peripheral veins, the process is the same so far as hemoptysis is concerned, though perhaps such emboli are more apt to be infectious and cause inflammation, suppuration, or gangrene.

SYMPTOMS.—It is not very rare to find at autopsy that blood has been poured out into the air passages when there were no symptoms of it during life. Such a hemorrhage may be of limited extent and have taken place gradually, or it may be very considerable, and have caused suffocation before any of it has appeared outside. This latter condition is seen more especially in apoplexy of the lungs.

As a rule, hemorrhage occurs in patients who are so sick that we are not unprepared for its occurrence. These are phthisical or cardiac cases mostly, though rarely hemorrhage occurs quite unexpectedly in people who appear to be in perfect health.

In many patients subjective symptoms precede a hemorrhage. They often say that they have a sensation of something warm rising up under the sternum, and then, with cough, blood appears. Exceptionally, they feel the sensation in the spot which is the source of hemorrhage. Others speak of an insipid, salty, or bloody taste in the mouth which causes hawking, and blood is expectorated. Some have a peculiar irritation which causes cough, or they complain of pressure in the head or oppression and fulness in the chest. Sometimes these complaints precede hemorrhage by several days, and are then to be considered as premonitory signs.

The quantity of blood varies very much. Sometimes it appears simply as small red spots or streaks in the sputum. In other cases it is pure blood or blood intimately mixed with the other constituents of the expectoration. It may be so abundant that in the course of twenty-four hours several pints of blood are lost. If the hemorrhage is rapid and copious, blood may stream from the mouth and nose and cause vomiting, so that blood and vomitus are mixed together, such a case being termed pneumorrhagia. Sometimes there is only a single attack of bloody expectoration. Often the hemorrhage is repeated several times at intervals of a quarter or half an hour, or several attacks may follow each other with periods of several days between them. An hæmoptysis dependent on malaria is described as recurring at certain hours of the day.

Sometimes the hemorrhage is uncontrollable, and death directly results from it.

It is characteristic of blood-tinged and pure blood sputum that the color of the blood is almost always bright red and arterial. Pure blood sputum is at the same time foamy, and, if there are any clots, air spaces are apt to be found in them on section, giving them a porous, sponge-like appearance. Such clots are therefore usually of a very light weight, though if the blood have settled in a cavity of some size and remained there for a time, it is frequently black in color and solid. The color of sputum intimately mixed with blood varies; it may be dark colored, as is sometimes seen in putrid bronchitis and gangrene of the lung. In hemorrhagic infarct, on the other hand, it is not rarely rusty like that seen in croupous pneumonia, the hemorrhage taking place slowly and in a limited space. Hæmoptysis may occur without our being able to detect the slightest change on physical examination. But if some alveoli have become filled with blood, fine moist râles may be heard under favorable circumstances. Dulness on percussion can only be detected when alveoli are filled with blood over a space four to

six centimetres in diameter and at least two centimetres thick, and percussion at the same time must be very gentle. If the focus is larger and includes bronchi of a tolerable size surrounded by alveoli filled with blood, we get physical signs of infiltrated lung—namely, increased vocal fremitus, dulness, and bronchial respiration—provided, of course, that the bronchi are free; if they are not, fremitus over such an area is lost and the respiratory murmur absent.

If fluid blood is in the bronchi, we get medium and coarse moist râles, which are loud when the focus is near the surface, and low and distant when deeper in.

When the hemorrhage is very profuse and rapid, asphyxia may result from stopping up large bronchi. The patient becomes deeply cyanosed, respiration is difficult, and the auxiliary respiratory muscles are called into play; the chest is drawn in over the affected area at each inspiration, or, at all events, is less distended than normal, and we feel no vocal fremitus and hear no respiratory murmur over it.

If the hæmoptysis has come from embolism, a chill usually precedes the bleeding by a few hours or days; sometimes faintness occurs.

The duration of the hemorrhage may vary. It may last but a very short time or continue for days, weeks, or even months.

Elevation of temperature is very apt to follow, which may be caused by an embolus in a branch of the pulmonary artery, resorption fever, an inflammatory process from infective emboli, or inflammation of the pleura dependent on hemorrhagic infarct from cardiac disease. Among the rare complications icterus is mentioned, which is apparently hæmatogenous, and in very profuse hæmoptysis anæmic systolic cardiac murmurs are heard.

Perfect recovery from hæmoptysis is not so very uncommon, though in the lay mind subjects of this trouble are doomed to speedy death. Hæmoptysis may kill the patient by suffocation or from loss of blood. In other cases inflammatory changes in the lungs occur in addition, as abscess, gangrene, or pleurisy, and bring about the fatal issue.

Diagnosis.—Hæmoptysis is not always easily recognized. It may be confounded with bleeding at the nose, throat, and gums. It sometimes happens that a slight nose-bleed occurs in the night during sleep, the blood flowing down into the pharynx or larynx, and on awakening is coughed up, giving the appearance of hæmoptysis. In all such cases careful inquiry should be made as to previous nose-bleeds or occasional occurrence of secretion from the nose streaked or mixed with blood, and the nose should be carefully inspected in front and behind with the mirror.

A careful inspection of the pharynx and gums, as a rule, will readily show if the blood came from there.

Hemorrhage from the larynx or trachea can usually be detected by the aid of the laryngoscope.

The differentiation between hæmoptysis and hæmatemesis, on the other hand, may be very difficult.

In hæmoptysis the blood is ejected by coughing, in hæmatemesis by vomiting. But in very profuse hæmoptysis simultaneous vomiting occurs not rarely, and, on the other hand, in copious hæmatemesis blood often flows into the larynx and is expelled by coughing. In

hæmoptysis the blood usually looks arterial and bright red, and is mixed with air bubbles, while in hæmatemesis, as a rule, it is black and clumpy. If large arteries, again, are eroded in the stomach, the gastric hemorrhage will show blood of an arterial character, while, on the contrary, in an hæmoptysis, where it has remained a long time in a cavity, it will be blackish red and coagulated. The blood in an hæmoptysis usually has an alkaline reaction, while in hæmatemesis it is acid, since it is mixed with acid gastric juice. If, however, the gastric hemorrhage is very considerable, it can readily happen that the gastric juice is not in sufficient amount to alter the normal reaction of the blood. We then see a fluid which is either neutral or just alkaline. Considerable admixture of food constituents favors the diagnosis of a gastric hemorrhage; absence of it favors pulmonary hemorrhage, though here, again, the difficulties first mentioned are encountered. The appearance of a black or bloody stool some time after the questionable hemorrhage is rather suggestive of hemorrhage from the stomach. The history given by the patient in hæmoptysis will point toward disease of the respiratory organs, in hæmatemesis to disease of the stomach. We must depend chiefly on the objective changes discoverable in the organs.

If the diagnosis of an hæmoptysis has been established, it must then be determined whether it is of bronchial or pulmonary origin—a thing which is not always possible, though the etiology is of great assistance in this matter. It is of relatively little consequence to determine whether the hemorrhage is arterial or capillary; which is fortunate, since it is very difficult or even impossible to decide the point in almost all cases.

It is of the greatest importance to know as soon as possible whether an hæmoptysis is of tuberculous origin or not, so that the detection of tubercle bacilli in the expectoration has great diagnostic worth. A microscopic examination of the sputum is necessary for the recognition of Gregarinosis, Distomiasis pulmonalis, and echinococcus of the lung, provided there are portions of echinococcus membrane or echinococcus hooklets in the sputum.

Prognosis.—The immediate prognosis in hæmoptysis is not unfavorable, as a rule. It may happen that a profuse hemorrhage may kill the patient directly by stopping up the bronchial tubes and causing asphyxia, or the patient may bleed to death; still, these are very unusual occurrences. It is only when hemoptysis depends on rupture of an aneurysm in a pulmonary cavity or on apoplexy of the lungs that the prognosis is unfavorable from the first. The ordinary attacks of hæmoptysis are apt to recur for a few days, and the sputum remains tinged or mixed with blood for some little time, but, as a rule, the hemorrhage ceases spontaneously. The ultimate result depends upon the cause of the hemorrhage and its amenability to treatment. If from the presence of tuberculosis, the stage at which the hemorrhage occurs governs the prognosis, that at a very early stage presenting some hopes of benefit to the underlying cause from treatment, while that which comes on at a very late stage is of course hopeless. The hemorrhage from an extensive gangrene of the lung is very apt to be repeated, profuse, and finally fatal, while that from a small area may be wholly recovered from.

Cancer and sarcoma give a fatal prognosis. The parasitic diseases are more favorable. The prognosis in hemorrhagic infarct depends on the condition of the heart and the source and nature of the embolus. The other forms of hæmoptysis are rarely fatal.

TREATMENT.—Prophylactic measures under certain circumstances may prevent the occurrence of hæmoptysis. Patients with acute or chronic inflammatory diseases of the respiratory organs should avoid exposure to cold and physical over-exertion. Severe irritative cough should be combated by narcotics. In cardiac disease a careful use of digitalis should be enjoined to avoid any possible loosening of thrombi, and in thrombosis of peripheral veins undue moving or handling of the extremities should be avoided for the same reason. In vicarious hæmoptysis efforts should be directed to bring on the normal flow of blood.

If called to a patient during hemorrhage, measures should be used to check it. The patient is to be sent to bed in a fresh, cool room; he is to be reassured and the anxiety of the people around him is to be allayed, and at the same time all muscular movements should be avoided and the patient should not talk. Any careful physical examination of the chest is to be postponed for some days, though auscultation of the breath sounds in the ordinary respiration is permissible, but percussion should by no means be practised, as it might easily cause the bleeding to recur.

The patient should take only liquid food, and that cold. An ice bag may be placed over the portion of the chest from which it is believed the blood comes, and small pieces of ice may be swallowed. Ergot by the mouth or subcutaneous injections of ergotin, 1 gr. or more, may be given morning and evening or oftener for a day or two. If there is much tendency to cough, morphine, $\frac{1}{12}$ gr., every three hours, or opium, $\frac{1}{2}$ gr., every two hours, should be given to allay it. Expectorants should only be used where there is danger of asphyxia by stopping up of the bronchial tubes through profuseness of the hemorrhage. Tracheotomy might be required to clear out the largest tubes or larynx.

Common table salt in doses of several teaspoonfuls dissolved in water has been swallowed with good effect, and is a harmless household remedy. Bromide of potassium in doses of 20 gr., three times daily, has been useful. Astringents, advised by all writers, are acetate of lead, 1 gr., with or without opium, $\frac{1}{2}$ gr.; tannic acid, 2 gr., every two hours; gallic acid, 20 to 30 gr., every two to four hours; alum; liquor ferri chloridi. Astringent and styptic inhalations are of questionable utility, if not positively harmful from the forced respiration which accompanies their use. Turpentine, ten to thirty drops on sugar or in emulsion every two to four hours, is said to be a valuable hæmostatic. The mineral acids are frequently used in ordinary repeated doses. Quinine, 10 to 15 gr., is advised where it is desirable to relieve congestion and in the malarial cases, and may be repeated within twenty-four hours. Aconite is also recommended for vascular relaxation. Hot external applications have sometimes proved efficacious when other means have failed, as mustard, very hot wet compresses, or poultices. Ligature with compresses of extremities has been in use since Hippocrates, and has its advocates at the present day. It should be so performed as to compress the large

veins, but not the arteries, in order that the blood may continue flowing into the limbs, while its return is obstructed. This simple means of reducing arterial pressure in the lungs should always be attempted in bad cases, for it can do no harm, and it very often has an immediate effect in causing absolute cessation of bleeding. The ligatures with compresses applied over the large veins of the arms and legs should be worn for twenty minutes or half an hour, and then removed very cautiously, one at a time.

The subsequent treatment of pulmonary hemorrhage is practically that of Simple Anæmia, to which the reader is referred. (See page 665.)

PNEUMONIA; BRONCHO-PNEUMONIA; CHRONIC FIBROUS PNEUMONIA.

By REGINALD H. FITZ, M. D.

PNEUMONIA.

Definition.—Pneumonia is an acute infectious disease due to the invasion of the lung by a variety of bacteria, oftenest by the diplococcus pneumoniæ, Fränkel's pneumococcus, and characterized by the production of a fibrinous inflammation of this organ pursuing a more or less typical course, and manifested by a variety of symptoms in part due to the absorption of toxins from the lung.

Although the anatomical seat of the inflammatory product is used to designate this disease, modern investigations tend to demonstrate that such localization chiefly represents the channel of admission of the bacterial cause, which exceptionally may enter the body through other ways. Pneumonia is thus analogous to typhoid fever, the lungs being conspicuously affected in the former, the intestine in the latter, while in both diseases associated disturbances of function and lesions of organs occur in remote parts of the body.

Various terms have been applied to pneumonia in accordance with the prevailing views of the importance of its localization, its products, and their distribution. When regarded as a primary inflammation of the lung it was called lung fever or pneumonitis. In view of the variation in its products fibrinous pneumonia was distinguished from catarrhal pneumonia, and the former was called also croupous, since in croup a fibrinous exudation in the larynx is often found. As more or less lung tissue was involved, lobar, lobular, and miliary pneumonias were differentiated. If the inflammation of the lung tissue immediately surrounded a bronchus, the condition was designated broncho-pneumonia. If the interstitial tissue of the lung was conspicuously affected, the condition was known as interstitial pneumonia, while from the fact that the pleura is often simultaneously involved the term pleuro-pneumonia was frequently applied. The classification here employed is based upon the character and situation of the anatomical products; hence fibrinous, croupous, or lobar pneumonia are regarded as synonymous with pneumonia, while catarrhal and lobular pneumonia and capillary bronchitis are considered as equivalent to broncho-pneumonia. The term fibrous pneumonia is preferred to interstitial pneumonia, since in this affection the fibrous tissue is often found in the alveoli. Hard-and-fast lines cannot be drawn between these anatomical varieties of pneumonia, since a fibrinous exudation may be found in lobar and lobular pneumonia, in broncho-pneumonic nodules, and may be present also in acute fibrous pneumonia.

Etiology.—Fibrinous pneumonia is a disease of frequent occurrence in various parts of the world. Holmsen estimates that in a single season at various times from 4 to 11 per cent. of the entire population of Norway have been under treatment for pneumonia. Barry states that from 3 to 4 per cent. of the patients in a number of hospitals in St. Petersburg were affected with pneumonia. It occurs nearly twice as often in the winter months as during the summer. It has been stated that nearly 7 per cent. of all deaths are due to pneumonia. Seitz in Munich, Delafield in New York, and Fränkel and Reiche in Hamburg found that the greatest number of cases occur in the spring months. The disease is found among men nearly four times as often as among women. It exists most frequently among young adults, although old men and infants are not spared. During the first five years of life a lobar pneumonia is considered usually as infrequent, the inflammations of the lung in infancy and early childhood being either broncho-pneumonia or lobular pneumonia, and the exudation more cellular than fibrinous. Southworth[1] states, however, that about one-third of the cases of pneumonia of the first two years of life show the characteristics of lobar pneumonia. It occurs more frequently among the weak and debilitated, whether from faulty hygienic surroundings, previous disease, the abuse of alcohol, or from old age. The robust and vigorous also may suffer, and in them the symptoms are more pronounced, a distinction thus being drawn between asthenic and sthenic pneumonia. Faulty hygienic surroundings may be found in poor ventilation and in bad drainage, while exposure to cold, formerly regarded as the chief cause, is now recognized to be of minor importance, and rather a predisposing than an exciting cause. Dwellers in malarial regions are frequent sufferers from atypical pneumonia, although, according to Marchiafava and Guarneri, the specific pneumococcus is present. Persons suffering from gout have been regarded as especially liable to pneumonia, but in 708 cases of acute pneumonia at St. Thomas's Hospital[2] a history of gout was present in but 1 of 565 cases terminating in recovery, while in 143 fatal cases evidence of gout was found after death only in 8. The importance of injury as an immediate cause of an attack of pneumonia is advocated by Litten,[3] who states that of 320 cases of pneumonia admitted into Frerich's wards, 4.4 per cent. were due to contusion. In 3 cases reported by Patterson[4] the disease began within twelve hours after the injury, and Burton[5] reports 2 cases, in both of which the symptoms rapidly followed the injury. It is probable that the above-mentioned conditions act as predisposing causes, diminishing the power of the individual to resist the activity of the invading cause. Mention may be made of the cases reported by Thomas and Ehrhardt of fibrinous pneumonia attributed to the inhalation of a powder composed of phosphoric acid and lime, acting as a chemical and mechanical irritant, the pneumo bacillus and pneumococcus being absent from the sputum and the lungs, although Enderlen[6] in 2 similar cases found the pneumococcus.

That pneumonia was not a local disease of the lung, but an infectious

[1] *New York Medical Journal*, 1894, lix. 134.
[2] *St. Thomas's Hospital Reports*, 1891, xix. 247.
[3] *Zeitschrift für klinische Medicin*, 1882, v. 26.
[4] *Lancet*, 1894, i. 136.
[5] *British Medical Journal*, 1893, 1425.
[6] *Klin. med. Woch.*, 1892, xxxix. 869.

disease characterized by local changes in the lung, was strongly advocated by Austin Flint, Jürgensen, and others.

The infectious nature of pneumonia has been suggested by its frequent typical course, the independence of general symptoms and pulmonary lesions, and the occurrence of epidemics and endemics. The epidemic occurrence of pneumonia has been long admitted. Halweed reports 50 cases of pneumonia in thirteen days in a village of 400 inhabitants. In 1888, in Middlesborough, England, a city of 40,000 inhabitants, there were 367 cases of pneumonia. Darlington[1] in the winter of 1886 treated 150 cases among laborers on the New Croton aqueduct who lived crowded together in close quarters. Townsend[2] states that of 643 cases of pneumonia among the poor in a certain section of Boston, 487 occurred in separate houses. Of 156 cases, 2 or more occurred in the same house. Flint stated that in more than 70 per cent. of all cases where pneumonia appeared in a house previously spared, it was proved that the person first attacked either immediately or a short time before had been in more or less intimate relation with a patient with pneumonia. Oliver[3] reports 3 cases in one family. The disease developed in the first patient while on a voyage from Antwerp. Death took place on the 3d of June. Three days later a second member of the family was taken sick and died at the end of nine days, and three days after the second patient fell ill a third was seized with pneumonia, but recovered. Evidence of this sort, which is of no infrequent occurrence, was sufficiently suggestive of a specific cause of pneumonia before bacteria in this disease were discovered. The history of the discovery of the pneumococcus and its relation to pneumonia recently has been admirably presented by Welch.[4] According to him, Eberth, Koch, and Friedländer found this organism in pneumonic lungs, and Leyden and Günther demonstrated its presence in fluid withdrawn during life from the diseased lung. In 1883, Friedländer cultivated a bacillus from the inflamed lung, but Talamon and A. Fränkel were the first to obtain cultures of the pneumococcus, and to show that the bacterium derived from pneumonia had pathogenic properties when inoculated. Sternberg and Pasteur several years before had found the pneumococcus in sputum, but were not aware of its relation to pneumonia. Sternberg and A. Fränkel identified this sputum coccus with the pneumonia coccus. According to Netter, this organism has been found in the mouths of about 20 per cent. of healthy individuals. Weichselbaum in the bacteriological examination of 94 cases of genuine lobar pneumonia concluded that the majority of cases of this disease are due to the diplococcus pneumoniæ. Although Fränkel has maintained that all cases of genuine acute lobar pneumonia are due to the diplococcus, this claim is opposed by numerous observers, who, although admitting it for the majority of cases, find other bacteria also concerned. Among these are Friedländer's bacillus, Pfeiffer's influenza bacillus, the streptococcus pyogenes, and the staphylococcus aureus. It is asserted also that the typhoid bacillus has been the cause of pneumonia occurring in the course of typhoid fever. Wassermann[5] during epi-

[1] *New York Medical Record*, 1888, xxxiv. 672.
[2] *Boston Medical and Surgical Journal*, 1890, cxvii. 291.
[3] *Lancet*, 1890, ii. 760.
[4] *Bulletin Johns Hopkins Hospital*, 1892, iii. 125.
[5] *Deutsche medicinische Wochenschrift*, 1893, xix. 1201.

demics of influenza has found Pfeiffer's bacillus exclusively in the sputum of pneumonia not to be distinguished from fibrinous pneumonia, and mentions that an influenzal pneumonia has been immediately followed by an attack of pneumonia attributed to the presence of the diplococcus pneumoniæ. Mason[1] reports 77 cases of pneumonia in 174 cases of influenza.

The diplococcus pneumoniæ or pneumococcus of Fränkel, sometimes called the lanceolate diplococcus from its frequent shape, is usually arranged in pairs, and is readily stained by aniline dyes. An important diagnostic characteristic is the presence of a capsule. It has been found not only in normal sputum and in the hepatized lung of pneumonia, but also in the milk of nursing mothers suffering from this disease, in the blood of pneumonic patients, and in that of infants born from parents with pneumonia. It has been found in the inflammatory exudation, in the numerous complications of pneumonia, as pleurisy, pericarditis, ulcerative endocarditis, peritonitis, meningitis, nephritis, tonsillitis, otitis, parotitis, arthritis, periarthritis, and in superficial and in deep abscesses. Many of these affections have been occasioned by the experimental introduction of the diplococcus pneumoniæ. Although Gamaleia and Emmerich have produced a fibrinous pneumonia in animals, most investigators regard such a result as exceptional. The diplococcus pneumoniæ at times is found, in the absence of pneumonia, in a variety of local inflammatory processes, especially in meningitis. A fibrinous pneumonia is therefore to be regarded merely as one of the results of the invasion of the body by this organism, and the efforts to produce fibrinous pneumonia by inhalation of the pneumococcus are usually negative, while its introduction into the bloodvessels produces a septicæmia.

Pathological Anatomy.—The characteristic changes to be found in pneumonia are seated in the lungs. They begin as an engorgement of the bloodvessels, followed by the presence in the alveoli and smaller bronchi of cells and fibrin, which in favorable cases are largely absorbed. These stages of the inflammatory process usually are spoken of as engorgement or congestion, hepatization, and resolution. In the same lung one portion may be in a state of engorgement, another in that of hepatization, while in another resolution may be taking place, or, what is more common, the appearances of two of these stages are to be seen in continuity. The anatomical changes affect one or more lobes of the lung, hence the name lobar pneumonia. They occur oftener in the right lung, while both lungs are affected in about one sixth of the cases. The lower lobe is diseased in about three fourths, and is alone diseased in about one half of the cases. The upper lobe is affected in about two fifths of the cases, being alone diseased or in conjunction with other lobes in about equal proportions.

In the stage of engorgement there is a combination of congestion and œdema. The lung is distended, heavy, moderately resistant, and dark red. On section a dark, somewhat viscid blood containing small air bubbles escapes. On microscopic examination the capillaries are injected, the epithelial cells swollen, and the alveoli contain an albuminous fluid in which are red and white blood corpuscles and desqua-

[1] *Boston Medical and Surgical Journal*, 1890, cxxii. 145.

mated granular epithelium. This stage usually lasts less than twenty-four hours, but may be continued several days.

In hepatization the appearances of the lung suggest that of the liver. The distended lung is heavy, and may be increased in weight three- or fourfold. It is friable, dense, non-crepitant, and pieces of it sink in water. The pleural surface is opaque and usually more or less abundantly covered with a fibrinous false membrane; therefore the terms pleuro-pneumonia or peri-pneumonia are applied. Frequently the surface is indented by the ribs. On section the color is either red or gray; hence red hepatization or gray hepatization is found, according to the duration of the process. Red hepatization corresponds to an earlier stage, and the color is due to the presence of a considerable quantity of blood, especially in the vessels. In gray hepatization there is less blood found, owing to the abundance of the exudation. The cut surface has a granular appearance from the presence of the fibrino-cellular exudation in the alveoli, from which, as casts of the same, the granules easily may be scraped. They are composed of fibrin, of red and white blood corpuscles, and of desquamated alveolar epithelium, and in gray hepatization these granules are larger and more opaque. In an emphysematous lung they are larger still; and the color of the cut surface is variegated with dark red or black spots when hemorrhage complicates, as is often the case in drunkards, or when excessive pigmentation of the lung pre-exists. An iron-rust color is present when pneumonia affects the lung in a state of chronic passive congestion. Fibrino-cellular plugs frequently are present in the bronchi, thus showing that a fibrinous or croupous bronchitis may exist wholly independent of any croupous (suffocative) symptoms. The interstitial tissue of the lung may be swollen and opaque from a cellular and fibrino-cellular exudation following the course of the lymphatics.

According to Ribbert,[1] the more abundant the fibrin the fewer the pneumococci, they being more numerous when the exudation is conspicuously cellular. In the pneumonia of elderly people and of those enfeebled by chronic disease the color of the lung is paler and the consistency is flabby, since the fibrinous exudation is less abundant and is associated with more fluid. Hepatization usually begins on the second day, and generally disappears within two or three days after the crisis has occurred. The stage of resolution is indicated by diminished resistance of the heavy lung, the color of which is more yellow, the cut surface less granular, and by the appearance on pressure of an opaque puriform fluid in which are cells in a state of granular and fatty degeneration and a granular detritus. If the lung is crushed between the fingers, the resistant pleura remains unbroken, but the spongy framework gives way, resulting in the formation of an irregular cavity in which the puriform fluid collects—an appearance often mistaken for abscess of the lung.

Variations in the anatomical appearances due to modifications in the quantity of blood and pigment have been mentioned. Limitations to the distribution of the exudation may occur; hence apical and central pneumonias are to be recognized. A lobular infiltration may exist also in fibrinous pneumonia, and offers an explanation of the milder course

[1] *Fortschritte der Medicin*, 1894, xii. 371.

and more rapid termination of certain cases of this affection. In wandering pneumonia a number of lobules in different portions of the lung are affected at different times, and the early and late stages of the inflammation thus may be found simultaneously in the same lung.

Although the fibrinous inflammation of the lung usually terminates in resolution, gangrene of the affected lobe sometimes occurs. This result is due to putrefactive organisms, which, easily admitted by way of the bronchi, find suitable opportunities for their development in the diseased lung, whose nutrition is arrested either entirely or in part. The gangrenous portion of the lung is indicated by an ill-defined cavity of large or small size containing a dark green offensive material easily washed away and exposing a shreddy wall.

The termination of pneumonia in pulmonary abscess is not infrequent, although it is maintained by many that the abscess is not a primary effect of pneumonia, but is due to the formation of an encapsulating membrane of granulation tissue around a gangrenous mass. The appearances of the lung in acute pneumonia, which are often mistaken for abscess, have been mentioned above. In rare instances the hepatized lung, instead of undergoing resolution, becomes transformed into a dense reddish gray, homogeneous mass containing little or no air, the further consideration of which result is to be found in the section on Fibrous Pneumonia (page 217).

Attention already has been called to a fibrinous inflammation of the smaller bronchi in which fibrino-cellular plugs or casts are then present, but at times the larger bronchi are similarly affected. More frequently a catarrhal bronchitis coexists, especially in the non-inflamed portions of the lung, in which event the mucous membrane is swollen and red. The lymphatic glands at the bifurcation of the trachea are enlarged, soft, and injected. The right side of the heart is usually distended with firmly clotted blood entangled among the trabeculæ. The spleen is frequently enlarged, soft, on section of a reddish gray color from hyperplasia of the pulp, the follicles and trabeculæ being indistinct. The association of such changes of the spleen with granular degeneration of the heart, liver, and kidneys has been long regarded as evidence of the infectious nature of the attack in which they are present. The pericarditis which occasionally complicates a pneumonia is more likely to occur when the left lung is affected. It is of a fibrinoserous type, and the exudation, sometimes abundant, usually contains the pneumococcus. Endocarditis is also an occasional complication, being either of the simple or ulcerative type, and diplococci have been found in the diseased valve. Inflammation of the pia mater, leptomeningitis, usually of the convexities of the brain, is a somewhat rare complication, and then often is associated with malignant endocarditis. In the meningeal exudation pneumococci have been found.

Symptoms.—In the consideration of the symptoms of acute pneumonia it is to be remembered that marked, even extreme, differences are to be found. On the one hand, the onset is sudden, the symptoms sharply defined, the course typical; on the other hand, the onset is gradual, the symptoms indefinite, and the course irregular. In one series of cases the symptoms point conspicuously to the disturbance in the function of the lungs, while in another series the pulmonary dis-

turbances occur as a complication of other diseases, as typhoid fever, malaria, or nephritis. Distinctions are thus drawn between the genuine, frank pneumonia and a secondary typhoid, asthenic, or other variety. Such differences in the clinical characteristics are associated with differences in the pathological anatomy and with variations in the bacteria present. Wassermann and Finkler, in particular, have recently presented the evidence in favor of accounting for such differences in the clinical characteristics by the prevalence of one or another form of bacterium. The diplococcus or pneumococcus is to be found in the large majority of cases of genuine fibrinous or croupous pneumonia, while staphylococci, streptococci, the typhoid bacillus, influenza bacillus, and still other varieties of bacteria are concerned in the secondary varieties.

Typical fibrinous pneumonia generally begins with a sudden chill, which may be preceded by a nasal or pharyngeal catarrh and slight malaise. Opinions vary as to the duration of the period of incubation; premonitory symptoms, according to most authorities, existing for two or three days before the onset of characteristic symptoms. The chill is mild or severe, being more prominent in the strong and vigorous, but its intensity is in no way proportional to the severity of the disease. It comes on unexpectedly, often while the patient is at work, perhaps while he is asleep, and may last half an hour or longer. As a rule, the severer the chill, the shorter its duration. The chill soon is followed by a rise of temperature and other symptoms characteristic of fever. Pain may follow immediately the chill, although it may not appear until later in the course of the disease, and may be even absent, especially in central pneumonia. The pain is stabbing on inspiration, and is commonly regarded as pleuritic. It is usually referred to the vicinity of the nipple corresponding to the seat of the inflamed lung. It may, however, be localized in remote points of the side or back. Dyspnœa is the next initial symptom of importance, in part due to the pain, in part to the demand for air occasioned by the congested condition of the lung, which is suggested by a frothy, bloody, viscid sputum. At this time the physical signs are indicative of the congested state of the lung, and numerous coarse and fine moist and dry râles are present, perhaps widely distributed, and to be heard even in parts of the lung which are not to become infiltrated. The stage of engorgement usually lasts from one to two days, and the pain and dyspnœa are often lessened as solidification of the lung takes place. During the subsequent course of the disease fever and dyspnœa are commonly the conspicuous features. The cheeks are flushed, the expression is anxious, the skin hot and dry, the pulse frequent, full, and strong. The respiration is rapid and superficial, often associated with groaning and interrupted by a short, painful cough. The sputum, often scanty, becomes viscid and rusty. The patient may be restless, mildly delirious, or, if alcoholic, actively delirious, requiring even restraint. There are headache, backache, loss of appetite, and thirst, scanty, high-colored urine, and constipation. The patient is weak and dislikes to be moved. This stage usually lasts three or four days. Between the fifth and eighth days, as a rule, resolution takes place, although it may occur both earlier and later. The temperature falls by crisis or lysis, during which the patient sweats profusely,

the cough becomes less painful, the respiration easier, while the general condition of the patient rapidly improves. The physical signs of hepatization continue several days, and exceptionally several weeks, after the fall of temperature has occurred.

The temperature rises, rapidly reaching its maximum, 104° or 105° F., at the close of the first day, or perhaps not until the second or third day. It usually remains near this point, with morning remissions and evening exacerbations of perhaps a degree. At times a longer fall takes place on the third or fourth day, sufficient to suggest a crisis, but to be immediately followed by a considerable rise; hence a pseudo-crisis. The critical fall of temperature, which may be preceded immediately by increased elevation, occurs oftenest on the seventh or eighth day, and occasionally on the ninth day, but sometimes after this date or exceptionally before the seventh day. According to Hawkins,[1] it takes place on or before the eighth day, more frequently in basic than in apical pneumonia. Its more general occurrence on the odd days is commonly admitted. The crisis is likely to take place in the night, and the temperature becomes normal or subnormal, usually in the course of a few hours. A subsequent rise of one or two degrees is not infrequent, but the temperature becomes again normal in about thirty-six hours. In other cases defervescence is by lysis, the temperature falling one or two degrees daily during three or four days, until it reaches the normal. The persistence of an elevated temperature after the crisis or lysis is suggestive of the presence of some complication, as pleurisy, gangrene, or abscess, or of an incipient chronic fibrous pneumonia. Pneumonia of the upper lobe may be accompanied by an unusually high range of temperature and with marked mental disturbance.

The respiration usually is between thirty and forty a minute, and may be much higher in children and in nervous persons. The increased frequency of respiration is out of proportion to the acceleration of the pulse, the normal ratio of the former to the latter being as one to four. In pneumonia, however, the ratio may be so increased as to be one to two or even less. The breathing is the more painful when inflammation of the pleura exists, and the pain is usually more severe during the earlier days of the disease, but may be slight or absent. The cough is commonly frequent and distressing, but may be wholly absent throughout, especially in feeble persons and in the central varieties of pneumonia. When present it is usually short and dry during hepatization, while it is often moist and accompanied with abundant secretion during engorgement and resolution, and is attributable chiefly to the associated bronchitis.

The sputum varies in its characteristics according to the stage of the disease. It is generally of moderate quantity, and sometimes may be wholly absent. At the outset it is a glairy mucus or a viscid, frothy, bloodstained fluid, according as it is due to a bronchial catarrh or to a pulmonary congestion. During hepatization it remains viscid, but is gelatinous, and is so tenacious that it adheres to the cup containing it when turned upside down. In color it is of various shades of red, especially the reddish yellow of iron rust, hence is called rusty. In severe cases the hemorrhagic element is so extreme that the color may resemble

[1] *Practitioner*, 1893, I. 434.

that of prune-juice. Nothnagel observed in certain cases a grass green color, which, according to Traube, is more often present in subacute pneumonia and in those cases ending in abscess. Von Jaksch found that this color was due to the transformation of hæmoglobin into bilirubin. On microscopic examination the sputum contains variously altered red blood corpuscles, leucocytes, and degenerated and pigmented alveolar epithelium. The pneumococcus and other varieties of bacteria are to be found, although the presence of the former in sputum is not necessarily indicative of the existence of pneumonia. It may contain fibrinous casts of the bronchioles, often visible to the naked eye when the sputum is diluted with water and spread in thin layer. During resolution the sputum becomes thick, yellow, opaque, and of a mucopurulent character.

The frequency of the pulse is closely related to the height of the fever. The pulse curve usually runs parallel to that of the temperature. The rapidity of the beat frequently is between 110 and 120, and is especially rapid in persons of nervous temperament. In children the pulse may be above 150 without giving any cause for anxiety, whereas in elderly people the frequency of the pulse may be below 100. At the crisis the pulse may fall 50 beats in the course of a few hours. During the early part of the disease the pulse is full and strong, while just before the crisis it may be small and compressible, irregular, and sometimes dicrotic.

Disturbances of the nervous system are frequent, especially in the old and young and in hard drinkers. Headache is common. A mild delirium, especially at night, is frequent in the course of a few days, but at times first appears as the crisis approaches. Even during convalescence attacks of delirium may occur. In general, severe cerebral symptoms are more likely to take place in connection with an unusually high range of temperature, such as is present often in apical pneumonia. In children the onset of the disease may be announced by convulsions, and delirium, perhaps violent, may follow. In such cases meningitis is often suspected until the physical examination shows the affection of the lungs. In drinkers an attack of delirium tremens, perhaps sufficiently severe to require restraint, frequently occurs in the course of the pneumonia.

The appetite is feeble and thirst is considerable. Vomiting takes place frequently in children. Constipation is the rule, although diarrhœa may be present, especially in children, early or late in the disease. The latter symptom is of graver import when of late occurrence.

Physical Examination.—The patient usually lies upon the back or on the affected side, and in case breathing is painful the head is raised and the body bent forward. The nostrils are expanded on inspiration. The cheeks, as a rule, are flushed. The lips and nose may be purple, and then are strongly contrasted with the pallor of the skin around the mouth. Jaundice is sometimes present; if early in the disease, it is attributable to associated gastro-duodenal catarrh or to passive congestion of the liver; if it occurs late, it may be due to the absorption of an exudation rich in red blood corpuscles. Febrile jaundice, according to Leube, should always excite a suspicion of pneumonia. Herpes either of the lips or nose, or of both, is present in nearly one third of the

cases, and usually about the third day. Sudamina are frequent when sweating is profuse.

On inspection of the chest there is less expansion of the affected side during inspiration, but the intercostal spaces are apparent. On palpation vocal fremitus is increased, and pleuritic friction sometimes may be felt, especially early in the disease. If abundant pleuritic effusion is present or if the bronchi are obstructed by considerable secretion, tactile and vocal fremitus are of doubtful recognition.

On percussion during the stage of congestion the resonance of the chest is not only undiminished, but is usually tympanitic. During hepatization there is rather dulness than flatness over the affected lobe; this sign is often first to be recognized in the subscapular region and toward the posterior axillary line, and usually is accompanied by a sensation of increased resistance on percussion. A high-pitched tympanitic note may be present if the solidified part of the lung is separated from the wall of the chest by an aërated portion, or if large bronchi distended with air are covered by a relatively thin layer of hepatized lung; these conditions are found especially in the upper lobe or when gaseous distention of the stomach is associated with pneumonia of the left lower lobe. Under these circumstances the pitch is higher when the mouth is open and lower when shut. The cracked-pot sound, higher or lower in pitch as the mouth is open or shut, usually is present when the upper lobe is infiltrated. Resonance appears in the dull area when the temperature becomes normal, although dulness may remain for days, even weeks, after the crisis and at a time when convalescence is progressing favorably.

On auscultation during the period of congestion coarse râles and the fine, moist, crepitant râle are to be heard, especially on inspiration, throughout both lungs. With the advance of solidification crepitant râles disappear, and the breathing becomes bronchial or tubular, perhaps associated with coarse râles. If the bronchus is obstructed, bronchial breathing may be arrested, but prolonged inspiration or an attack of coughing may cause a displacement of the obstructing secretion with a return of the tubular breathing. Bronchophony, sometimes ægophony, is to be heard also over the hepatized area in which bronchial breathing is present, but may be absent temporarily when the bronchus is obstructed. With the occurrence of resolution the tubular breathing and bronchophony are replaced by moist râles (crepitus redux), which are usually coarser than the crepitant râle of the congested lung, and are to be heard on inspiration. In central pneumonia there may be neither dulness nor bronchial breathing, and crepitant râles may be absent. Bronchophony, however, is to be heard.

The area of cardiac dulness may be increased to the right, especially when the heart's action is labored in virtue of the severity of the disease. The second pulmonic sound is usually accentuated.

The spleen and liver are often found enlarged.

Especial importance is to be attached to the examination of the blood. The red blood corpuscles may be somewhat diminished, and the blood plates are said to be increased. The especial change is the presence of leucocytosis, which progresses with the advance of the dis-

ease. According to Ewing,[1] it ranges between 20,000 and 32,000, and Kidd[2] has found the number of leucocytes as high as 100,000 in a particularly virulent case. The degree of leucocytosis is usually higher the more severe the disease, yet a moderate leucocytosis is no indication that the case will pursue a favorable course. Ewing observed no case of recovery in which the leucocytosis was below 14,000, and although so low a number of leucocytes in acute pneumonia is a sign of bad omen, the exceptions to this experience are sufficiently numerous to make this feature rather of relative than of absolute value in prognosis. A return to the normal number of leucocytes takes place during resolution. According to Monti and Berggrün,[3] slight but persistent diminution in the number of leucocytes at a time when improvement may be expected announces a speedy occurrence of the crisis. An increased leucocytosis, even in the absence of physical signs of extending infiltration, indicates an increase of the pulmonary invasion.

The urine is scanty, acid, of high color, and of high specific gravity. The chlorides almost completely disappear during the height of the disease, but return after the crisis, at which time the quantity of urine becomes increased. Albuminuria occurs in more than one third of the cases, and its presence is associated with hyaline casts, which are more abundant the larger the quantity of albumin. The urine usually becomes freed from both in the course of a week after defervescence. A brick-dust sediment is frequent during the height of the fever, and is most abundant at the time of the crisis. The diazo-reaction is sometimes present, and, according to von Jaksch, the presence of peptone is indicative of commencing resolution.

VARIETIES OF PNEUMONIA.—Attention already has been called to the possible importance of a various bacterial etiology in explaining the differing characteristics of cases of pneumonia, and it has been suggested that a satisfactory explanation for the variation in the clinical course of this disease thus might arise. The primary typical lobar pneumonia has been distinguished from the secondary variety occurring in typhoid fever, erysipelas, septicæmia, and in various other infectious processes characterized by considerable fever and great prostration. The secondary varieties of pneumonia have been called asthenic pneumonia in virtue of their progress being associated with extreme prostration, a lower range of temperature, greater feebleness of pulse, more mental disturbance, and frequent splenic enlargement. In these cases a considerable albuminuria is more constant and casts more abundant, serious complications are more common, the mortality is higher, and a flabby hepatization of the lung is likely to be found. This series includes cases of so-called wandering pneumonia, some of which perhaps result from the direct extension of a facial erysipelas to the respiratory tract. The term wandering pneumonia is applied also to those cases in which the hepatization affects one portion of the lung after another, the regions earlier involved undergoing resolution, while those later attacked are either congested or hepatized, and the fever either remains constant or shows remissions and exacerbations, the latter corresponding with the inflammation of fresh portions of the lung. The progress of these cases

[1] *New York Medical Journal*, 1893, lviii. 713. [2] *Practitioner*, 1884, liii. 182.
[3] *Archiv für Kinderheilkunde*, 1893-94, xvii. 1.

sometimes suggests that of acute tubercular affection of the lung, but Wassermann in one such found only pure cultures of streptococci in the lungs, and the patient showed no reaction to the tuberculin test.

The pneumonia occurring in malarial regions is said at times to present characteristics differing from those of typical pneumonia and resembling those of asthenic pneumonia, with intervals of freedom from the symptoms lasting often but a few hours. Hadgi-Costa[1] states of these intermittent or post-paludal pneumonias that they are of insidious onset, classical symptoms are slight or absent, organic and functional disturbances of the abdominal organs are frequent, and that grave nervous and adynamic symptoms predominate. Resolution is slow, suppuration frequent, and the mortality great. Marchiafava and Guarneri have found the pneumococcus in malarial pneumonia.

Typhoid pneumonia is a term often used in a double sense. On the one hand, it represents the asthenic type of pneumonia with conspicuous typhoidal symptoms, especially stupor, dry tongue, swollen abdomen, and diarrhœa; on the other hand, it is used to indicate the occurrence of pneumonia in typhoid fever. In the former case the presence of streptococci or staphylococci is probable, while in the latter the inflammation of the lung may be due to the presence of the typhoid bacillus.

The term bilious pneumonia, as is above stated, represents the occurrence of jaundice in pneumonia. The association is more frequent in the asthenic, typhoidal, or septic varieties of pneumonia.

Ephemeral pneumonia is applied to the presence of the symptoms and signs of pneumonia ending in the course of forty-eight hours, the stage of hepatization not having been reached. In abortive pneumonia the stage of hepatization may be present, but resolution immediately follows, and convalescence occurs on the third or fourth day. Apical and central pneumonias are also to be recognized, the former term being applied when the infiltration begins in the upper lobes. In apical pneumonia the symptoms often are severe, grave nervous disturbance is present (hence the term cerebral pneumonia), and the course is likely to be protracted, perhaps from delayed absorption through the narrowed lymphatics which are so often found at the apex of the lung. The existence of a central pneumonia is oftener inferred than demonstrated. The rational signs, as chill, fever, cough, and pain, are present, but the physical signs are lacking, with the exception perhaps of bronchophony. Relapsing pneumonia is said to occur when, soon after the crisis, a return of the fever takes place with symptoms and signs of pneumonia, the latter localized either in the lobe previously affected or in another lobe.

The pneumonia of children, elderly persons, and of those addicted to the abuse of alcohol presents certain features deserving of especial consideration. The frequent occurrence of lobar pneumonia in young children is a matter of comparatively recent recognition. This in part is due to the frequent occurrence in them of broncho-pneumonia and to the absence in children of the symptoms characteristic of pneumonia in the adult. Townsend[2] recently has called attention to this variation in the symptoms. He states that sudden vomiting and occasional convulsions more frequently announce the onset of pneumonia in a child than does a chill. The child fails to localize the pain, or refers this symptom

[1] *Revue de Médecine*, 1891, xi. 927. [2] *Archives of Pediatrics*, 1889, vi. 148.

to the abdomen or to some other part of the body. There is little or no expectoration, and rusty sputum is rare. The physical signs of consolidation are slow in appearing, and crepitant râles are infrequent. The occurrence of an apical pneumonia as the cause of delirium and stupor should be thought of in the differential diagnosis of the acute inflammatory affections of young children. Townsend emphasizes particularly the favorable prognosis of the uncomplicated acute lobar pneumonia in children, and has prepared a table of 1138 cases which shows a mortality of about 2 per cent. In senile pneumonia, on the contrary, the onset of the attack is often obscure, and the fever, pain, cough, and expectoration are slight. The signs of consolidation may be but little apparent, and resolution takes place but slowly. In the pneumonia of drinkers the symptoms of the pulmonary affection may be overlooked from the frequency of delirium tremens. Thoracic pain and fever may be slight, cough and dyspnœa may be inconspicuous, although the sputa may be abundant and of a prune-juice color. The delirium may be noisy and restless or low and muttering. The patient's condition is often such that mechanical restraint becomes necessary. A sudden fatal termination from cardiac failure is not infrequent.

Complications.—Bronchitis is a frequent accompaniment of pneumonia, and attention has been called to the presence of fibrinous plugs in the smaller bronchi. A catarrhal bronchitis affecting the larger bronchi is relatively frequent, and is the cause of the numerous coarse râles to be heard in the early and late stages of pneumonia. Pleurisy also is a frequent, almost constant, complication, except in cases of central pneumonia. There may be extensive pleurisy of the upper and lower lobes, while the lower lobe alone is affected with pneumonia. The pleurisy is usually more fibrinous than serous, and pneumococci have been found repeatedly in the exudation. Thick masses of fibrin or large quantities of serum may intervene between the lung and the chest wall, markedly obscuring the physical signs of solidification. If a diagnosis between pleurisy and pneumonia is thus made doubtful, the use of the aspirator may be essential in the differentiation. The exudation is sometimes purulent, according to Mazzotti, in about 1 per cent. of the cases. Empyema is to be suspected from the persistence of dulness, the absence of respiratory sounds, the continuance of the fever after a critical fall of temperature, and the presence of the signs of resolution in the upper portions of the affected lobe. A continued leucocytosis is also important evidence.

Pericarditis is more likely to occur when the pneumonia affects the left lung, and slight degrees of this complication are so frequent that repeated examinations for a pericardial rub should be made. The exudation is fibrinous or serous, usually contains pneumococci, and is sometimes so abundant as to seriously interfere with the movements of the heart. In the severer varieties of pericarditis the fluid may be seropurulent. Endocarditis is an occasional complication of pneumonia, and, according to Osler, nearly 25 per cent. of the cases of malignant endocarditis tabulated by him occurred in pneumonia. The aortic and mitral valves are usually concerned, although Netter found in 82 cases of endocarditis in pneumonia that the valves of the right side of the heart were diseased in 12 cases. Pneumococci repeatedly have been

found in the vegetations. An intact valve or a previously diseased valve may be the seat of the inflammation. Arterial embolism in various parts of the body thus may arise as a complication of pneumonia.

Meningitis occasionally occurs, especially in connection with endocarditis. The exudation of sporadic meningitis arising independently of pneumonia often contains pneumococci, and in epidemic cerebro-spinal meningitis pneumonia frequently occurs as a complication. Peripheral neuritis is to be mentioned as one of the rarer complications of acute pneumonia, and Reilley has reported the occurrence of symmetrical gangrene as a sequence of pneumonia. Arthritis is an infrequent complication, but pneumococci have been found in the resulting exudation. Parotitis has been observed, and pneumococci have been found in the purulent exudation. Prioleau[1] reports suppurating orchitis as a sequence of pneumonia, the pus containing pneumococci. Hemorrhages from the mucous membrane, especially of the nose and intestine, may occur, particularly during the later stages of the disease. Hæmaturia also may take place, especially when acute nephritis is present as a complication. Hemorrhage from the genital tract may be profuse when miscarriage occurs in pneumonia.

Diagnosis.—The symptoms and signs of acute pneumonia are usually so characteristic that the diagnosis is attended with but little difficulty. The stage of congestion may be simulated by the dyspnœa, cyanosis, frothy, bloody expectoration, and the moist râles of acute œdema. The absence of fever, the general distribution of the râles, their temporary character, and the evidence of associated cardiac disease suffice for the differential diagnosis. The sudden onset, localized pain, rapid breathing, cough, and rusty sputum, when accompanied by the physical signs of solidification of the lung, leave but little room for doubt. When the diagnosis is difficult, it usually results from neglect in examining the upper lobes of the lung, or from the delayed appearance of the characteristic signs of solidification, or from the existence of central pneumonia, in which relatively normal lung overlies the diseased portion. In such cases the examination of the blood is of great importance, since leucocytosis usually is present. When in doubt, daily search should be made for the physical characteristics of this disease, since signs of solidification may be absent for several days. If the physical signs of pneumonia are slow of development, it may be necessary to wait for several days before the diagnosis is made. Especial difficulty may arise in the case of children, in whom the cerebral symptoms, especially convulsions, delirium, or stupor, may be so marked at an early stage as to suggest the existence of meningitis. In delirium tremens the fever and rapid respiration, without a corresponding increase of pulse rate, should lead to the examination of the lungs, which will reveal the existence of the pneumonia if present. Acute tuberculosis at times is mistaken for pneumonia, although in the former the febrile course is more irregular and rusty sputa are lacking. (See Vol. I. pp. 753, 754.) The previous history of the patient, the discovery of the bacilli of tuberculosis in the sputum, the absence of a leucocytosis, and the more protracted course of the disease may enable eventually the differential diagnosis to be made. Acute pleurisy with abundant exudation

[1] *Limousin méd.*, 1895, xix. 117.

may be mistaken for pneumonia, since dulness, bronchial breathing, bronchophony, and crepitation are common to both. The onset of pleurisy is usually more gradual, the expectoration less abundant and free from blood. The bronchophony and tubular breathing of pneumonia are more marked near the lower level of dulness, the reverse being the case in pleurisy, and tactile fremitus is more constantly increased in pneumonia, but is diminished in pleurisy. In pneumonia there is no displacement of the heart or liver. In cases of persistent doubt the aspirator should be employed with a view to determine the presence or absence of fluid.

PROGNOSIS.—According to the statistics of Fränkel and Reiche, from 16 to 23 per cent. of the cases of pneumonia prove fatal. The mortality in individual epidemics varies, however, within wide limits, in accordance with a variety of conditions. Pneumonia in children is usually recovered from. In the aged the mortality is high, while in adults enfeebled by disease or alcoholic habits the prognosis is serious. At St. Thomas's Hospital[1] of 61 cases in which alcoholism existed, 31 died. Typical fibrinous pneumonia is more readily recovered from than the asthenic cases, in which the symptoms of toxæmia, especially delirium, rapid respiration, and feeble pulse, are present. A few cases may prove fatal before the lung is solidified, but the majority die in the stage of red hepatization. Death usually takes place shortly before the crisis is due, while a few patients, even in the absence of complications, die soon after this event. The prognosis in the individual case is more grave in proportion to the extent of lung involved, double pneumonia being more serious than pneumonia of one lung or of one lobe. Apical pneumonias are generally considered as having a higher mortality than basal pneumonias. Pneumonia occurring in emphysema, heart disease, nephritis, or pregnancy has a grave prognosis, and the more advanced the pregnancy the more likely are miscarriage and death. Unfavorable symptoms in adults are a progressively increasing frequency of pulse and respiration, tracheal râles persisting despite efforts at coughing, prune-juice sputum, stertorous breathing, low delirium, and muscular tremor. The prognostic significance of leucocytosis has already been referred to. Ewing has stated that recovery did not occur when the number of leucocytes was below 14,000, that absence of leucocytosis was almost invariably fatal, and that moderate leucocytosis when the symptoms were severe was unfavorable, while a marked leucocytosis does not assure a favorable course. The occurrence of pericarditis renders the prognosis more serious. The immediate causes of death in pneumonia are various. Most important is the toxæmia due to the pneumotoxin produced by the pneumococcus, which occasions cardiac incompetency, probably through its effect on the nervous system. In consequence of the weakened heart and of the hepatization of the lung, and from the rapid and superficial character of the respiration, the blood becomes insufficiently aërated and the patient cyanotic. The pulse is rapid, irregular, and weak. The respiration is noisy from the presence of bronchial and tracheal râles, and the patient becomes drowsy, comatose, and dies. Bollinger[2] attaches especial importance as

[1] *St. Thomas's Hospital Reports*, 1891, xix. 247.
[2] *Münch. med. Woch.*, 1895, xlii. 745.

a cause of death to the quantity of the exudation which represents a corresponding loss of important constituents of the blood.

Treatment.—The recognition of pneumonia as an infectious disease due to the action of specific bacteria led the Klemperers[1] and others to seek for a healing agent in the blood of inoculated animals. They succeeded in immunizing rabbits against the effects of the pneumococcus. The blood serum of these animals was injected into other rabbits infected with the pneumococcus, with the effect of neutralizing the toxalbumin of the pneumococci and relieving the symptoms. Serum obtained after the crisis from cases of pneumonia acted in like manner. Five to 10 c.c. of serum from immunized rabbits were injected into a number of patients with pneumonia with doubtful benefit. Neisser injected 130 c.c. of serum obtained from patients after the crisis into 3 patients with pneumonia, in 2 of whom a critical fall of temperature immediately took place, and in the third a pseudo-crisis occurred. Audeoud[2] injected 2 to 3 c.cm. of the serum from convalescent patients, and crisis followed on the fourth day of the disease within thirteen hours after the injection. A control injection of blood serum from a patient not having pneumonia was followed by a crisis in forty-eight hours. At present no further claim is made for the sero-therapy of pneumonia than that it may produce the earlier occurrence of the crisis. It seems probable that an antipneumotoxin appears in the blood at the time of the crisis, and is efficacious in producing the latter and in neutralizing the effects of the pneumococcus, although some days may be required for the removal of the exudation. Issaef[3] maintains that the serum of vaccinated rabbits possesses no antitoxic power and is incapable of attenuating the virulence of the pneumococcus.

Since leucocytosis is absent or slight in the severer cases of pneumonia, von Jaksch has suggested that the use of such agents as pilocarpine, antipyrine, antifebrin, and nuclein, which increase the number of leucocytes, might be beneficial in the treatment of pneumonia.

It is probable, judging from the results of the analysis made by Townsend and Coolidge[4] of 1000 cases treated at the Massachusetts General Hospital between 1822 and 1889, that the mortality of pneumonia has been influenced but little by any special form of therapy. It is therefore important in treating an individual case to maintain the patient's strength to the utmost possible extent and to relieve symptoms. Danger usually arises from enfeebled action of the heart and deficient aëration of the blood. All treatment, therefore, which tends to weaken the action of the heart should be considered as harmful. Mild cases of pneumonia may require no medication whatsoever.

The essential requisite for severe cases of pneumonia is the sustaining treatment. The treatment of pneumonia by venesection has probably destroyed many more lives than it has benefited. It is doubtful if it gives other aid than temporary relief to a patient struggling for breath during the stage of engorgement. At such a time, if the patient is robust, with flushed face, rapid and noisy breathing, frothy and bloody sputa, the loss of a pint of blood may be followed by relief to the

[1] *Berl. klin. Woch.*, 1891, xxviii. 833.
[2] *Revue médicale de la Suisse Rom.*, 1893, xiii. 130.
[3] *Ann. de l' Inst. Pasteur*, 1893, vii. 260.
[4] *Tr. Am. Climat. Ass.*, 1889, vi. 22.

dyspnœa, headache, or drowsiness, but death may ensue in the course of twenty-four hours. If venesection be employed, its use should be limited to the first forty-eight hours of the disease, before solidification has taken place. Even then it may be dangerous by the removal of blood from a person in whom, as emphasized by Bollinger, a practical intra-visceral hemorrhage to the extent of from 2 to 4 pints is about to take place.

Of other treatment employed in former times, that with veratrum viride still has its advocates, who recommend it as a means of temporary relief during the stage of congestion or throughout the disease. The indications for its use are those permitting venesection, to which it is inferior in offering immediate relief. Its effect in lowering the force of the heart is not desirable during the stage of hepatization, and there is no evidence that it shortens the duration of the disease.

The patient should be placed in a well-ventilated room kept moderately cool. Except in cases of extreme elevation of temperature but little attempt should be made to lower the body heat, except by occasional cold sponging when the temperature is above 103° F. Extreme measures, especially cold baths and a wet pack, are of doubtful value. Applications of ice bags to the chest are warmly advocated by Mays[1] of Philadelphia, who reports a mortality of 3 to 4 per cent. in cases thus treated. Relief to pain, cough, and dyspnœa is claimed, and it is stated that râles appear in the course of a few hours in the hepatized region. The use of the antipyretic drugs is undesirable from their effect in weakening the heart.

During the first few days of pneumonia but little other treatment is necessary than that designed to relieve the patient's discomfort. The pain, which is aggravated by deep inspiration and cough, is often considerably lessened by encircling the chest with a broad cotton bandage made comfortably tight by means of straps and buckles. The pain is also to be relieved by local applications of heat, cold, sinapisms, or leeches. The last may be recommended especially if there is severe pain during the stage of engorgement. Blisters are to be avoided from the risk of prolonged discomfort following their use. The pain may be so severe that the use of morphine is required. Some preparation of opium is also necessary to relieve painful or harassing cough when present. For this purpose Dover's powder, 3 to 5 grains, either in liquid or solid form, is frequently given, and may be continued, when indicated, throughout the disease. Its use is obviously uncalled for if the patient is drowsy or the larger bronchi are obstructed with secretion. Cough mixtures other than Dover's powder are unnecessary and of but little use. There may be neither cough nor secretion from the lung during the disease, the inflammatory product eventually being absorbed. The viscid pneumonic sputa largely arise in the smaller bronchi. Their evacuation is favored by abundant liquids, preferably the milder saline waters charged with carbonic acid gas, as soda water, Apollinaris, Vichy, and the like. Headache, wakefulness, and delirium, if present, are often relieved, if due to febrile excitement, by cold compresses or an ice bag upon the head or by cold sponging of the body, and the use of opium, trional, or hyoscin hydrobromate, especially at

[1] *Medical News*, 1893, lxiv. 681; *ibid.*, 1894, lxv. 403.

night. Large doses of alcohol and mechanical restraint may be necessary if delirium tremens occurs. During the latter half of the disease, as the pulse increases in rapidity and diminishes in tension and the respiration becomes more superficial, the use of alcohol, strychnine, atropine, and nitro-glycerin is indicated. Although opinions vary as to the value of alcohol, it is to be employed obviously in patients accustomed to its use. Its benefit is likely to be greater if given to patients unaccustomed to its use when threatening weakness of the circulation and respiration precede or dangerous collapse follows the crisis. In such cases alcohol is to be given freely in the form preferred by the patient, if choice exists, until improvement in circulation and respiration indicates that the object is accomplished. Of late years sulphate of strychnine has been freely used in doses of about $\frac{1}{50}$ grain, either subcutaneously or by the mouth. The indications for its administration are the same as those requiring alcohol. The concurrent employment of these remedies may permit smaller doses of alcohol to be taken than if this remedy alone is given. If benefit is to result from strychnine, it must be experienced within half an hour after a subcutaneous administration, and its use may be continued at intervals of two or three hours while the patient's condition is critical. Sulphate of atropine subcutaneously, in doses of $\frac{1}{100}$ grain, is warmly advocated by some as strengthening the action of the heart and improving the character of the respiration. If relief does not follow two or three doses at intervals of three or four hours, its further use should be discontinued. Nitro-glycerin in doses equivalent to 1 minim of the spirit of glonoin may be given at intervals of an hour, especially when the cardiac weakness is associated with increased tension of the pulse. Such administration, if productive of relief, should take place at frequent intervals, since it is rapidly eliminated. Havem[1] recommends inhalations of nitrite of amyl at frequent intervals throughout the disease for the relief of the symptoms referable to the lungs. He advises that 15 drops be placed on a compress which is to be held over the nose and mouth, and deep inspirations drawn. As much as 50 drops thus may be used, when beneficial, in the course of five hours.

Of late years inhalations of oxygen sometimes mixed with nitrous monoxide have been used freely, and often with immediate though, perhaps, but temporary relief, especially to the dyspnœa and cyanosis. The pulse may be lowered somewhat and improvement in the mental condition take place. Its administration requires frequent repetition, and patients react differently toward its use. Agreeable to some, to others it is objectionable. There is no reason to suppose that it shortens the course of the disease or diminishes the mortality. It may be administered as well during the stage of congestion as during the critical period preceding resolution. Its use is often satisfactory to the friends of the patient even when no other obvious benefit arises. Aromatic spirits of ammonia or sweet spirits of nitre, the latter indicated from the similarity of its action to that of nitro-glycerin, may be used throughout the disease, particularly in those cases where some medication is deemed expedient even if of doubtful value. German writers warmly recommend the use of musk when a patient is in a state of collapse. Its dis-

[1] *Nouv. rémèdes*, 1895, xi. 481.

agreeable odor to many and its inferior value to the other stimulants mentioned render its employment limited.

The administration of digitalis in pneumonia was recommended first for the purpose of checking the fever. Later it was largely employed in ordinary doses to diminish the frequency of the pulse. Its failure to shorten the duration of the disease and to diminish its mortality, its doubtful benefit in giving immediate relief, the frequent gastric disturbance following its use, its slow elimination, and the thought in the minds of many physicians that it may have rather injured than benefited the patient, have resulted in its disuse. At the best it is to be regarded as of doubtful value, though preferably to be employed in cases of pneumonia occurring in the course of chronic cardiac disease, especially mitral stenosis, and in those in which excessive frequency of the pulse exists. Strophanthus is a safer though less efficient cardiac stimulant, has no cumulative action, and may be discontinued without further ill effects if toxic symptoms arise. Petresco of Bucharest[1] of late years has advocated the use of large doses of digitalis in the proportion of 60 to 90 grains of the powder to 6 ounces of water, to be taken daily in doses of a tablespoonful every hour, or 4 or 5 mgm. of digitalin, each of which is the equivalent of 15 grains of powdered digitalis, hypodermically, each day, stating that such doses are tolerated by adults, and that there is no risk of poisoning. He has repeatedly observed the pulse to fall from 120 beats to 40 beats per minute, and the temperature from 104° to 96.8° F., and claims to have arrested the disease in four to five days. In 1890 the mortality in 816 cases under this treatment is stated to have been 2.6 per cent. Fikl[2] treated 60 cases of pneumonia, 46 being lobar and 14 lobular, during a period of fifteen months by the Petresco method, although using but 45 grains of digitalis daily. All recovered, although of 44 cases otherwise treated in a previous year 7 died. The course of the disease was more prolonged than in Petresco's observations. Vomiting, delirium, and mania occurred in some of the patients, and were regarded as symptoms of poisoning. Lépine[3] used digitalin, 3 mgm. in the morning and 1 to 2 mgm. in the afternoon, in 40 cases, with a favorable effect on the heart. Masius agrees that a drachm of digitalis may be taken in twenty-four hours without inconvenience, and states that the dangers of cardiac weakness are thereby surely and quickly prevented. In a very grave case of pneumonia almost in the death agony he gave from 120 to 150 grains of digitalis in twenty-four hours with brilliant success. Bloch[4] is a recent advocate of this method of treatment. Havas,[5] on the contrary, treated 3 cases by Petresco's method without any effect on the pneumonia. The patients complained of nausea, vomiting, faintness, and of specks before the eyes. The pulse became irregular and there was dilatation of the pupil. According to Lépine,[6] Löwenthal gave 45 to 60 grains of digitalis daily, without notable modification in the course of the disease, and the weakness of the patient increased. Lépine has injected 20 to

[1] *Revue de Médecine*, 1893, xiii. 199. [2] *Wien. med. Woch.*, 1891, xli. 1033.
[3] *La Semaine médicale*, 1892, xii. 21.
[4] *Wratsch*, 1894; *Centralblatt für klin. Med.*, 1895, xvi. 189.
[5] *Pester Medico-chirug. Presse*, 1894; *Centralblatt für klin. Med.*, 1894, xv. 832.
[6] Loc. cit.

26 c.c. of a solution of corrosive sublimate (1 : 40,000) into the lung near the periphery of the infiltration. As a result of this treatment he states that the symptoms improved, the course of the fever was shortened, and no harm resulted.

The diet should be largely of milk, broth, beef-juice, and eggs when well borne. In general the patient's preferences are to be followed. The especial treatment of the complications which arise is the same as if they occurred independently of the pneumonia, and is considered under their respective titles.

BRONCHO-PNEUMONIA (LOBULAR PNEUMONIA; CAPILLARY BRONCHITIS; CATARRHAL PNEUMONIA).

DEFINITION.—A circumscribed inflammation of the lungs, usually multiple, and generally secondary to bronchitis.

ETIOLOGY.—Broncho-pneumonia is a disease most frequently found in the early and late years of life and in weakened and debilitated persons. It prevails during the winter months, and exposure to cold is often considered an exciting cause, although it is to be regarded rather as a favoring factor, since broncho-pneumonia often occurs without any such exposure. The immediate cause is to be found in the inhalation of foreign material, the effects of which are dependent upon its quality and quantity and also upon the minuteness of its subdivision. Such material, when inhaled, produces an acute bronchitis, affecting the larger or smaller bronchi as the particles are large or small. If the larger bronchi are primarily inflamed, the process tends to be continued into the smaller bronchi; hence the term capillary bronchitis or bronchiolitis, which is practically synonymous with broncho-pneumonia. Such foreign material may be particles of food, perhaps introduced through a tube in the feeding of the insane or debilitated, or may be regurgitated from the stomach and inhaled through the larynx during anæsthesia. The inhalation of particles of food likewise may follow obstructed closure of the glottis, either from local disease of the larynx or from interference with the innervation of the vocal cords by aneurysmal or other tumors or by chronic disease of the brain or spinal cord. Broncho-pneumonia may be caused by the inhalation of blood, as in cut-throat, tracheotomy, or from hemorrhage into the respiratory tract otherwise occasioned. The products of acute or chronic disease affecting the larynx or pharynx may be inhaled as in diphtheria, typhoid fever, ulcers, and tumors of the pharynx and larynx. The inhalation of particles of dust in the various trades and of irritating gases also may be an excitant of broncho-pneumonia. The most frequent cause is the inhalation of the minutest particles—viz. infectious bacteria. The occurrence of broncho-pneumonia thus is explained in those diseases in which a catarrhal bronchitis is a customary lesion, as the exanthemata, especially measles, also in whooping cough, influenza, and diphtheria. The epidemic occurrence of broncho-pneumonia likewise is thus explained, and the course of the broncho-pneumonia may be modified by the nature of the bacteria, as in tuberculous broncho-pneumonia. Evidence of

the infectious origin of many cases of broncho-pneumonia is the presence of numerous bacteria, either alone or in combination in the inflammatory products. Among such are the pneumococci, which, according to Neumann, are found in the majority of cases of broncho-pneumonia in children; the streptococcus pyogenes, which abounds in the broncho-pneumonia of diphtheria and of erysipelas; staphylococci, the bacillus diphtheriæ, the influenza bacillus, Friedländer's pneumonia bacillus, and still other varieties of bacteria likewise are to be found. The conspicuous importance of the bacillus tuberculosis in the production of tubercular broncho-pneumonia has been considered in the article on Tuberculosis (Vol. I. p. 777). Broncho-pneumonia also occurs in a variety of infectious diseases, in which the complicating bronchitis is rather a secondary process in virtue of extreme prostration than a primary manifestation of the disease. The existence of broncho-pneumonia in certain cases of typhoid fever, dysentery, and in the traumatic and puerperal infections is thus accounted for, the weakened circulation and respiration favoring bronchial congestion and increased secretion, but preventing elimination. The occurrence of broncho-pneumonia is thus explained also in debilitating chronic diseases, as rickets, cancer, and nephritis.

PATHOLOGICAL ANATOMY.—The characteristic anatomical changes are due to the presence of leucocytes and alveolar epithelium with occasional red blood corpuscles, abundant serum, and more or less fibrin. These are present in the bronchioles, alveoli, and groups of alveoli, the surrounding fibrinous tissue being infiltrated with leucocytes. Thus nodules of various size and shape are formed, and are distributed throughout the lungs or are limited to certain portions, notably the posterior portions of the lower lobes in broncho-pneumonia from inhalation of coarse foreign bodies. The appearances vary in accordance not only with the distribution of the lesions, but with their duration and their immediate cause. The broncho-pneumonia of tuberculosis, for instance, often assumes a miliary character and presents a caseous appearance, while the lesions following the inhalation of food may be of a more lobular shape and rapidly tend to become gangrenous.

The lungs usually are found distended and injected, and do not readily collapse, especially when numerous bronchi are obstructed. Rounded or polygonal patches of a red color and sometimes elevated may be seen beneath the overlying pleura. The latter is often ecchymosed, opaque, and is covered often with a delicate fibrinous membrane. On palpation larger or smaller nodules are to be felt in the interior of the lung. On section of the lungs the broncho-pneumonic nodules appear as flattened or rounded, slightly elevated, homogeneous, usually reddish gray or dark red patches. Pressure upon them causes the escape of a drop or two of more or less opaque, often yellow, viscid fluid from the section of the obstructed bronchus traversing the nodule. At times on longitudinal section of the central bronchus its cavity is found filled with the inflammatory product or with a recognizable foreign body, as particles of food. The shape of the nodule is rounded or irregular, in the latter case often lobular. The smallest nodules are usually globular in virtue of the spreading of the inflammation to the alveoli immediately surrounding the affected bronchiole. The lobular nodules are large, since their shape is due to the extension of the bronchitis to the peripheral

bronchioles with their alveolar terminations. The lobular shape is due also to the frequent occurrence of broncho-pneumonia as a secondary condition in a collapsed portion of the lung. The collapse or atelectasis results from obstruction of the bronchus by secretion and displacement of the air beyond the point of obstruction. (See Atelectasis, Vol. II. p. 234.) Inflammation of the collapsed portion of the lung often, though not necessarily, follows. The broncho-pneumonic nodules are frequently surrounded by emphysematous alveoli, which are hyperdistended from the admission of an excess of air through neighboring unobstructed bronchi. They may be separated by considerable intervals of normal or injected lung, or be closely approximated and grouped in a grape-like cluster along the bronchial stem in one or many lobules of the lung. The intervening alveoli may be collapsed and their walls injected, perhaps œdematous. Extensive portions of the lobe, even the entire lobe, thus may become solidified. When the inhalation of food is the cause of a broncho-pneumonia, gangrene of the affected portion is likely to occur and rapidly to progress. The nodules then are soft, of a greenish color, and become shreddy as the destruction of tissue advances.

The previous existence of a broncho-pneumonia often is indicated by the presence of cicatricial fibrous tissue either in the form of few or many large or small nodules. Such fibrous scars may be wedge-shaped, extending toward the surface of the lung, which is usually depressed and adherent to the wall of the chest. These fibrous nodules usually are pigmented, and the larger may contain numerous yellow specks (from fatty degeneration of the alveolar epithelium) imbedded in a translucent gray fibrous tissue—a condition known as gelatinous œdema or slaty induration according to the duration of the process. It represents one of the several varieties of chronic fibrous pneumonia. The especial characteristic of tuberculous broncho-pneumonia is the necrosis of the nodule, which then presents the familiar cheesy appearance.

Symptoms.—Broncho-pneumonia offers no such sharply defined characteristic grouping of symptoms as is to be found in genuine fibrinous pneumonia. The disturbances are due largely to mechanical obstruction of numerous bronchioles occurring as a primary or secondary condition in the course of a variety of diseases; hence the symptoms in a primary broncho-pneumonia are indicative of a capillary bronchitis or bronchiolitis, while those of a secondary broncho-pneumonia may represent merely a modification in the character of the symptoms of the existing disease. In the consideration of the symptoms those of tuberculous broncho-pneumonia will be disregarded, since this subject has been fully treated in the article on Tuberculosis (Vol. I. p. 779). Primary broncho-pneumonia other than that of tubercular origin is almost invariably an acute process. Secondary broncho-pneumonia usually develops gradually and unexpectedly in the course of the disease of which it represents a complication. The symptoms of a bronchitis of the larger tubes generally precede, and the invasion of the smaller bronchi and the extension to the bronchioles and groups of alveoli is so gradual that the existence of the broncho-pneumonia is often overlooked.

Circumscribed broncho-pneumonia when limited to a part of the lung, especially to the apices, is usually of a tubercular character, but

a limited broncho-pneumonia of non-tubercular origin may be found in any portion of the lung. The symptoms of especial significance are fever, dyspnœa, and cough. The temperature rises to the vicinity of 102° or 103° F., and continues thus elevated without typical characteristics. If recovery takes place, the temperature gradually falls until convalescence is established. The higher and more prolonged elevations of temperature are indicative of a more extensive distribution of the lesions. The pulse and respiration become proportionately increased, the former rising to 150 or upward, and the latter to 60 or upward, especially in children. There is thus less disproportion in the ratio of pulse to respiration than is the case in fibrinous pneumonia. Dyspnœa is also present, and the accessory respiratory muscles are brought into prominent use. The nostrils are dilated, the intercostal spaces and the epigastrium are depressed, and the abdominal rectus is contracted. The respiration is frequently irregular and noisy from the presence of coarse râles in the trachea or bronchi, and the expiration is characterized by moans or grunts. The respiratory efforts may be so violent that rupture of the lung tissue occurs, and an interstitial emphysema results which may become extended to the subcutaneous tissue of the neck and chest. Cough exists from the outset, and may occur in paroxysms, often in children resulting in vomiting. When pleuritic pain is associated, the cough is suppressed and muffled to avoid the pain consequent upon taking a long breath. The speech is short, and the patient unwilling to talk through fear of inducing pain or cough. The sputum is viscid and streaked with blood, and usually is scanty in children, by whom it is generally swallowed, since the patient is either unwilling or unable to expectorate it. Percussion at the outset gives evidence of but little change, the pulmonary resonance perhaps becoming somewhat more tympanitic than normal. During the subsequent course of the disease dulness may occur, especially over the lower lobes behind and near the spine. On auscultation fine moist râles are to be heard associated with moist and dry râles of a coarser character. The former are more frequently to be found in the lower lobes, and are evidence of the disease in the bronchioles and alveoli, while the coarse râles are due to the associated bronchitis. The breathing is rarely tubular, unless numerous nodules are present in the vicinity of large bronchi, and bronchophony is usually lacking. The urine is scanty and high colored, and contains a trace of albumin. In the subsequent course of the disease improvement may take place, with lowering of the temperature, diminution of the cough and dyspnœa, and return of the appetite, the patient recovering by resolution in the course of a fortnight. A relapse, however, may occur, terminating favorably at the end of a few days or resulting in the development of graver symptoms. On the other hand, if the temperature remains elevated and the dyspnœa persists, the skin, at first dusky, becomes livid, the respiration is more noisy, with tracheal râles, the patient appears drowsy, convulsions may take place, especially in children, and then death occurs. In such cases abscesses or gangrene may be found in the lungs.

A protracted convalescence from broncho-pneumonia is observed especially in those cases in which extensive atelectasis is associated with broncho-pneumonic nodules. Slow absorption of the inflammatory prod-

uct takes place, and persistent cough, emaciation, and debility may continue for months. Eventually the collapsed lung becomes distended, and the health of the patient may be wholly restored.

Although isolated patches of pleuritic inflammation are frequent, any considerable liquid exudation is rare. Pericarditis, endocarditis, and meningitis, the complications of greater or less frequency in fibrinous pneumonia, are rare in broncho-pneumonia. Tuberculosis and broncho-pneumonia are frequently associated. The former may be the cause of the broncho-pneumonia, or foci of broncho-pneumonia may become tuberculous, as is shown in the frequent termination of measles in tuberculosis by the invasion of the broncho-pneumonic nodules in the former disease by tubercle bacilli.

Diagnosis.—The diagnosis of broncho-pneumonia is a matter rather of inference than of demonstration. The physical signs are those indicative of movable secretion, not of accumulated exudation in the minuter air passages. It is only when extensive broncho-pneumonia, associated with atelectasis or œdema and congestion, exists that dulness, bronchial breathing, and bronchial voice occur, and these are found more especially in the lower lobes near the spine, as in hypostatic broncho-pneumonia combined with atelectasis. The diagnosis largely depends upon the evidence of a broncholitis, as indicated by increased elevation of temperature, rapid breathing, dusky skin, and subcrepitant râles in the sequence of a bronchitis in childhood or in old age, and depending upon an obvious cause or occurring in the diseases previously mentioned, especially in measles and whooping cough, and the constitutional disturbances apparently being disproportionate to the local lesion.

Broncho-pneumonia is to be differentiated from lobar pneumonia by its more frequent occurrence at the extremes of life, by the presence of the signs of a capillary bronchitis or of a hypostatic infiltration instead of a lobar solidification, usually in both lungs instead of being limited to a single lobe, also by a less abrupt onset and a more prolonged course. The sputum of broncho-pneumonia is muco-purulent, while that of fibrinous pneumonia is rusty. The distinction between a simple and a tuberculous broncho-pneumonia is usually difficult, especially when the broncho-pneumonia is limited to a small portion of the lung. The physical and rational signs may be the same, but the primary simple broncho-pneumonia may become tuberculous, or broncho-pneumonia may be tuberculous from the beginning. Most important in the differential diagnosis is the evidence of tuberculous processes elsewhere, and the examination of the sputum or vomit, in case the pulmonary secretions are swallowed, for tubercle bacilli. Failure to find these does not wholly remove doubt. Persistence of fever and the sharply localized presence of signs of an exudation in the bronchioles or alveoli create a strong suspicion of the presence of the tubercular process.

Prognosis.—Broncho-pneumonia is regarded usually as presenting a high rate of mortality, from one half to two thirds of the cases being reported as fatal. This high rate of mortality is due largely to the fact that the cases generally tabulated are those of children, in whom the broncho-pneumonia is secondary to measles, whooping cough, or diphtheria, while the milder cases or those of unknown origin, especially in adults, are not included. The prognosis, therefore, varies largely in ac-

cordance with the etiology of the disease and the time of life at which it occurs. Broncho-pneumonia following the inhalation of food usually proves fatal. Its occurrence in diphtheria also usually ends fatally. Its presence in measles is far less serious, except in infants, while its development in the course of a capillary bronchitis of unknown or doubtful origin has a mortality which varies largely in accordance with the extent of the bronchitis; the more widely distributed the latter the greater the mortality.

The average mortality from extensive broncho-pneumonia probably lies between 30 and 50 per cent. Unfavorable signs are persistent dyspnœa and high fever, irregular respiration or Cheyne-Stokes breathing, delirium, convulsions, or somnolence, especially if the disease has existed for some days.

TREATMENT.—It is to be remembered that broncho-pneumonia usually occurs as the result of the extension of a bronchitis into the smaller bronchi, and with relative frequency in the course of certain diseases. It is therefore important to place the child with bronchitis, measles, or whooping cough, and the elderly person with bronchitis, under the most favorable conditions to promote convalescence and recovery. The special indications in the treatment of broncho-pneumonia are to check extreme elevation of temperature, to promote ease in breathing, to stimulate circulation and respiration enfeebled by asphyxia, and to offer a suitable diet.

It is customary, particularly in the broncho-pneumonia of children, to prescribe some saline preparation, especially spirits of nitrous ether, under the name of fever mixture, or to administer drop doses of tincture of aconite every few hours, with the object of lowering the temperature and soothing the patient. Young children rebel at such medication, and it is found often unproductive of relief in them or in their elders. Of late years the antipyretic drugs, especially phenacetin and lactophenin, have been largely employed in appropriate dose according to the age of the patient, particularly early in the disease, and often afford temporary comfort. The same object may be accomplished by frequent spongings with cold water, which is more especially indicated in the high fever of the later stages of the disease, since the lowering of the temperature thus accomplished is likely to be accompanied by relief to the disturbances of the nervous system and improvement in the circulation. Ease to the dyspnœa often is promoted, as in fibrinous pneumonia, by the application of a jacket of linen, cotton, or woollen, which may be agreeably tightened by means of tapes or straps and buckles. If local pleuritic pain is a source of discomfort, a bag containing ice or hot water, according to the preference of the patient, is to be applied locally. When such pain is so situated that local treatment is inconveniently administered, the use of some preparation of opium is indicated. Such opiates may be included in a cough mixture, which, especially in children, is the more likely to be taken the more palatable and less bulky it is; hence cough mixtures containing ipecacuanha, tartar emetic, or chloride of ammonium, so frequently advised, are not to be insisted upon. Opiated troches and sweetened opiated mixtures are more readily taken, and should be carefully administered, with the recognition that blunting of the sensibility leads to diminished evacuation of catarrhal material

from the bronchi. Frequent small doses of Dover's powder either in liquid or solid form, are generally commended as an efficient, if not a palatable, combination.

Most important in the removal from the bronchi of the inflammatory products is abundant liquid, which should be presented in the form of water in such agreeable form as may promote its use as food and drink; hence water plain or effervescing, cream-of-tartar water, lemonade, or ginger ale are to be taken freely. The air of the room should be moderately moist. Measures to prevent impending suffocation eventually become necessary. The removal of secretion from the trachea and bronchi may require the occasional use of emetics, such as tepid water, mustard water, or ipecacuanha. The value of cold sponging has already been referred to. The administration of strychnine and the use of digitalis, strophanthus, or citrate of caffeine are indicated. Carbonate of ammonium also is useful, and should be given at intervals of two or three hours for a number of days. Alcohol in some form is to be employed, and inhalations of oxygen may prove of temporary benefit. The diet should be largely of milk, broths, and eggs, with the addition of oysters, chickens, or steak when possible.

CHRONIC FIBROUS PNEUMONIA.

A PATHOLOGICAL formation of fibrous tissue in the lung proceeds from the bronchi and bloodvessels, the interlobular tissue, and the pleural covering. It takes place under a variety of conditions, and is usually a secondary result of the processes concerned. According as these processes are localized or diffused, so does the distribution of this new-formed tissue vary. A localized formation of cicatricial fibrous tissue repairs more or less extensive destruction of the lung, whether occasioned by injury, abscess, gangrene, or necrosis. Thus, after recovery from abscess or gangrene of the lung in acute pneumonia the loss of pulmonary substance is indicated by a fibrous cicatrix. The collapsed portion of the lung occurring in the course of bronchitis or broncho-pneumonia may be represented, if the affected portion of the lung is not again aërated, by a wedge-shaped fibrous scar. An extensive formation of fibrous tissue takes place in the course of tuberculosis, whether circumscribed or diffused, and represents a tendency to the healing of this condition as well as a line of demarcation restricting its advance. The absorbed gumma is represented by a fibrous nodule. The encroachment of encysted parasites, of actinomyces, of aneurysmal or neoplastic tumors, is controlled or checked by fibrous thickening of the lung in their immediate vicinity. Such localized conditions are to be regarded as a part of the phenomena of the diseased process concerned, rather beneficial than injurious.

The diffused new formation of fibrous tissue proceeding from the vicinity of the bronchi is the essential eventual characteristic of the morbid changes due to a chronic bronchitis involving few or many of the larger bronchi of one or of both lungs. This is the condition to

which, when extensive, Corrigan applied the term *cirrhosis of the lung*, from the analogy of the results to those occurring in fibrous hepatitis. The growth of fibrous tissue extends into the lung from the bronchial wall and follows the course of the bronchi. Thus is formed a series of arborescent fibrous bands radiating outward from the root of the lung. Dilatation of the bronchi, obliteration of the bronchioles, and emphysema of numerous alveoli are associated. In consequence of the destruction of numerous pulmonary capillaries during the progress of these changes increased resistance is offered to the flow of blood through the lungs, and hypertrophy and dilatation of the right ventricle of the heart occur. In virtue of these lesions of the lung and heart the symptoms at the outset are those of chronic bronchitis, followed later by those of bronchiectasis, and these in turn by evidence of passive venous congestion and defective aëration of the blood. The usual cause for such alterations is the inhalation of dust in their respective trades by the laborers exposed to coal-dust, by millers, stone-workers, steel-polishers, and the like. The detailed consideration of this subject is therefore to be found in the articles on Pneumonokoniosis and Bronchiectasis (pages 244 and 157).

Multiple circumscribed nodules of fibrous pneumonia are one of the results of chronic bronchitis and of broncho-pneumonia, independent in origin of the inhalation diseases of the various trades. Such results are especially likely to occur when the bronchitis is of tubercular origin. The especial consideration of this variety of fibrous pneumonia is therefore to be found in connection with the subject of Pulmonary Tuberculosis (Vol. I. p. 804).

Diffused fibrous pneumonia also represents an occasional event in the history of chronic pleurisy, especially of empyema. The term *pleurogenic fibrous pneumonia* is applied to this condition, since the growth of fibrous tissue extends from the surface of the lung toward the root. The affected lung is contracted, dense, and firm, and forms a flattened cake-like mass adherent along the spine to the upper and posterior portion of the affected half of the chest. The corresponding portion of the thorax becomes flattened and distorted; compensatory emphysema of the other lung takes place, with enlargement of the corresponding half of the thorax and displacement of the hypertrophied and dilated heart. Passive venous congestion and defective aëration of the blood are also a secondary result of this variety of fibrous pneumonia. The further consideration of the subject will be found in the article on Pleurisy (page 275).

In the limited and somewhat arbitrary sense the *genuine chronic fibrous pneumonia* is that variety usually described as representing one of the rare terminations of acute fibrinous pneumonia. According to Marchand,[1] it was recognized by Laennec as presenting similar characteristics to those of acute fibrinous pneumonia, and with difficulty discriminated from it. Stokes and Forbes also were familiar with a chronic pneumonia which they regarded as the result of the incomplete resolution of the acute variety.

ETIOLOGY.—But little is known concerning the conditions which determine the origin of genuine chronic fibrous pneumonia. Marchand

[1] *Virchow's Archiv*, 1880, lxxxii. 317.

suggests that a previous attack of pneumonia may leave behind extensive induration and pleuritic adhesions, which, by preventing absorption, may act as an important predisposing cause for the subsequent termination in induration of acute fibrinous pneumonia. He attributes importance also to the presence of an increased supply of blood to the lung from the chest wall, which is promoted by vascularized pleural adhesions. It is possible also that this variety of pneumonia may prove from the outset to be of a different bacterial nature from the common forms of acute fibrinous pneumonia. According to Marchand, the patients in whom this affection of the lung is found are alcoholic, badly nourished, and exposed to unwholesome hygienic surroundings.

Pathological Anatomy.—The characteristic appearances may be present within three weeks of the origin of the disease. The affected portion of the lung is distended, although, according to Virchow, not to the same degree as in fibrous hepatization. It is dense and heavy, notably tough and resistant. The pleuræ are thickened and adherent. On section the surface is smooth or slightly granular, shining, translucent, and of a pale, grayish red color. When the cut surface is scraped with the knife a yellow fluid is removed in which comparatively little blood is present, while fibrinous plugs, even in the third week of the disease, are absent. To this condition the term carnification is applied. At a later stage of the disease the color of the lung is more gray than red, and numerous yellow specks are to be seen in the translucent tissue. The peribronchial, perivascular, and interstitial fibrous tissue is thickened. The bronchi contain an opaque fluid of a cream-like consistency. On microscopic examination the alveolar walls are thickened, and the interstitial tissue often is infiltrated with leucocytes. The alveoli contain fibrillated, coherent casts, simulating those found in fibrinous hepatization, but composed of a granulation tissue in which injected capillaries are to be found. Acute pericarditis may complicate the process. It is probable that a more or less extensive increase of the fibrous tissue of the lung permanently results, and it may be that some of the cases of Corrigan's cirrhosis or fibroid phthisis thus arise. The clinical history of these cases suggests that the function of many of the alveoli eventually may be restored.

Symptoms.—The disease begins as an acute fibrinous pneumonia, and the temperature may become normal by crisis or lysis at the end of ten days. In other cases the temperature is 100° or 101° F., perhaps with slight intermissions, for a period of weeks. Rapid breathing and cough continue. The sputum becomes sero-purulent. The appetite improves. The pulse is 100 or upward and the urine is high colored. Dulness of the affected portion of the lung persists. Bronchial breathing and bronchophony may remain or disappear, and numerous coarse and fine moist and dry râles usually are to be heard. In the course of weeks or months, in cases not terminating fatally, the fever gradually disappears, the cough lessens, the strength of the patient improves, but a gradual retraction of the chest takes place. The shrunken chest remains permanent, but dulness may disappear and the respiration become broncho-vesicular. Chronic cough, shortness of breath, perhaps occasional cyanosis and œdema, are of likely occurrence from compensatory hypertrophy of the right ventricle.

Heller[1] describes a congenital interstitial pneumonia due to syphilis affecting symmetrically the whole of both lungs. The lungs are distended, dense, of a grayish red color. The increased interstitial tissue forms a coarse meshwork producing a narrowing of the alveoli. Pleural and pericardial ecchymoses, also hypertrophy of the right ventricle, are frequent. He considers that this affection, beginning during fœtal life, may produce the death of the child at birth or continue into adult life. Children affected with this disease are considered especially predisposed to bronchitis and pleurisy.

DIAGNOSIS.—In the early stages chronic fibrous pneumonia is with difficulty to be differentiated from the delayed resolution of acute fibrinous pneumonia. The latter condition is relatively common, but the general condition of the patients decidedly improves, although dulness, bronchial breathing, and bronchophony persist for several weeks. The continuance of the fever and the gradual retraction of the thorax, with the persistence of the signs of solidification, are the significant characteristics of chronic fibrous pneumonia.

PROGNOSIS.—Although eventual recovery from this disease occurs, the after effects remain throughout life. Chronic or recurrent bronchitis is likely to be present, and the copious muco-purulent sputum of bronchiectasis may be formed. The greater the deformity of the thorax, the more likely is hypertrophy of the right ventricle to occur, with displacement of the heart, and of the liver when the right half of the thorax is affected. Finally, signs of failing compensation arise which may prove the immediate cause of the patient's death.

TREATMENT.—In the early stages of chronic fibrous pneumonia attention to the nutrition of the patient is of the utmost importance. That absorption of some or many of the fibrous casts in the alveoli takes place seems probable from the extensive aëration of the diseased lung which sometimes occurs. In such cases doubt may be expressed as to the nature of the process, but the intimacy of connection of the blood-vessels in this granulation tissue with those of the lungs is so slight that atrophy, if not necrosis, of the granulation is favored. Solidification of the lung from probable fibrous pneumonia has disappeared during six months' residence in a high and dry altitude, and such climatic treatment is advisable as would be suitable for the early stages of pulmonary tuberculosis. During the subsequent life of the patient especial care is to be exercised with regard to exposure to cold and dampness or to excessive strain of a necessarily enfeebled heart. The further treatment is, therefore, that suitable for cases of chronic bronchitis or of failing cardiac compensation. (Compare pp. 141, 401.)

[1] *Beiträge zur pathologischen Anatomie und experimentellen Pathologie zur Feier von Zenker's Jubiläum.*

EMPHYSEMA; ATELECTASIS; ŒDEMA OF THE LUNG; ABSCESS OF THE LUNG; GANGRENE OF THE LUNG; PULMONARY EMBOLISM; PNEUMONOKONIOSIS.

By WILLIAM WHITWORTH GANNETT, M. D.

EMPHYSEMA.

Definitions.—The term "emphysema," when applied to the lung, refers to one of two widely different conditions. With the prefix "interstitial" a collection of air bubbles in the lymph spaces of the connective tissue is meant, due to rupture of the lung substance and the establishment of a direct communication between alveoli and the spaces in the interstitial tissue or stroma of the lung. With the prefix "vesicular" a substantive change is meant, associated with over-distention of the alveolar walls and enlargement of the alveolar spaces, which may go no further or which may be associated with atrophy and disappearance of the alveolar walls and vascular changes to be described later.

Interstitial emphysema is of anatomical rather than of clinical interest, though pneumo-thorax may occasionally result from it. It occurs in disease associated with violent paroxysms of cough, as whooping cough, and, while not to be diagnosticated by clinical examination, and not giving rise to symptoms, yet is readily apparent at the autopsy from a beaded appearance, like a string of pearls, following the course of the interlobular connective tissue, due, as stated, to air bubbles in the lymph spaces. In the majority of cases its occurrence is of no importance, for, unless death results from another cause, the air is quickly reabsorbed.

Vesicular emphysema is, for convenience, subdivided into three forms:

(1) Vicarious or compensatory;
(2) Senile;
(3) Vesicular emphysema proper.

(1) By vicarious emphysema is meant simply an enlargement of the alveolar spaces and over-distention of the alveolar walls, usually without substantive changes in the walls. It occurs in lungs which are already affected by some pathological process, rendering part of the organs impervious to air. A greater intra-alveolar pressure, therefore, results in those parts of the lung in which the air can freely circulate, and stretching of the alveoli follows. It may be permanent or transitory, according to the character of the underlying disease, whether

death results or recovery takes place. The original disease is of so much greater consequence that, clinically, the emphysema is insignificant.

The acute and chronic forms of pneumonia, atelectasis, and pleurisy are the affections with which this form of emphysema is usually associated.

(2) Senile emphysema is essentially an atrophic process, without enlargement of the lung as a whole, but characterized by special changes to be described later.

(3) Vesicular, substantive, or hypertrophic emphysema is associated with an enlargement of the lung, so far as its bulk is concerned, but with marked dilatation of the alveolar spaces, atrophy of the walls, and fusion of contiguous alveoli; so that, although the lung appears larger, yet there has been an extensive obstruction of lung substance.

Etiology.—Consideration of the underlying etiological factors has occupied writers from the time of Laennec, but it is now generally agreed that increased expiratory pressure is the main cause of the changes. In certain cases it is quite possible that congenital lack of resistance in the alveolar walls is a predisposing factor, Louis having observed the disease in children, and its occurrence in successive generations of the same family has been noted. Imperfect development of the elastic fibres must be the condition in these cases.

The part played by increased expiratory effort is readily understood if one considers the effect produced by closing or partly closing the glottis and forcibly expelling the air with the help of the expiratory muscles, such as occurs in coughing. The pressure within the alveoli is much increased, and the tendency to forcibly distend their walls is present, unless there is a counter-pressure brought to bear upon the outside. Such a counter-pressure is afforded in the lower half of the lung, this part being covered by the accessory muscles of expiration; but the upper half is not so protected, consequently the alveolar walls yield and enlargement of the alveoli results. If the cough is frequently repeated, as occurs in chronic bronchitis, the recurring increase of internal pressure causes a gradual yielding of the walls and a permanent dilatation both of infundibula and alveoli, and an associated thinning and atrophy of the inter-alveolar septa.

The essential change in emphysema is the diminution, and finally almost entire disappearance, of the elastic fibres in the alveolar walls.

A comprehension of the part these elastic fibres play in normal respiration is necessary in order to understand the main symptoms due to their absence. In the normal individual, during inspiration, the lung plays a passive part. The enlargement of the cavity of the chest by descent of the diaphragm and raising of the ribs by the inspiratory muscles, creating, as it does, a negative pressure, causes the air to enter, and so distend the lung as a whole, the elastic fibres in the alveolar walls being put upon the stretch very much as a rubber band may be stretched between the fingers. In this case the rubber band plays a passive part. But in the expiratory act the lung plays an active part, as the energy stored up in the stretched elastic fibres by contraction forces out the air, or rather a considerable part of it, and the lung as a whole becomes less voluminous.

Now, it is plain that if this elastic tissue is lessened or absent, the lung will either retract less well or not at all, unless helped by the accessory muscles of expiration. In other words, while inspiration may be performed fairly well, expiration is not only difficult and labored on the part of the individual, but is also much prolonged. Normally, expiration is about one third of the length of inspiration, but in emphysema it may be four times as long; that is, increased twelve fold. In watching a patient with emphysema one gets the impression that the individual has but one object in life—namely, to get the air out of the lungs.

Owing to the lack of this elasticity of the lung the tendency naturally is for the chest to assume more and more the position of inspiration and the shape characteristic of inspiration.

Vesicular Emphysema.

Etiology.—Vesicular emphysema is essentially a disease of middle and late life, though occasionally seen in children and young adults. Men are far more commonly affected than women.

Occupation is a causative factor in certain cases, for the disease occurs not infrequently in those who blow wind instruments, in glass-blowers, and in those engaged in occupations involving lifting, the act being associated with closure of the glottis, contraction of the abdominal muscles, and so increased intra-alveolar pressure.

In the majority of cases, however, emphysema is associated with bronchitis, and especially with that form of bronchitis which from lack of secretion has been called dry bronchitis. The cough in this form may be severe.

There has been much discussion as to the priority of the emphysema or the bronchitis. There can be little doubt that in the majority of cases the bronchitis is the primary affection, the emphysema secondary to it. The point most in favor of this idea is that, inasmuch as the mechanical origin plays the more important part in the production of emphysema as compared with a developmental cause, a prolonged cough, with the resulting increase of intra-alveolar pressure in expiration, is necessary as a starting-point of the disease.

It is well known that a dry bronchitis is likely to be associated with emphysema, while a bronchitis with much secretion is apt to result in bronchial dilatation of the alveoli. Recklinghausen used to explain this difference in result on the ground that in moist bronchitis the alveoli were protected by the secretion in the bronchioles from the increased pressure during cough due to the expiratory effort made with a closed glottis, all the pressure coming upon the bronchi, and in time leading to their dilatation; whereas without this protecting secretion the alveolar walls had to withstand this increased pressure and emphysema resulted.

Occasionally there are other causes of increased expiratory pressure than that of cough, such as a narrowing in the air passages, especially the larger ones, due to presence or pressure of new growths. This also may lead to emphysema.

Pathological Anatomy.—The tissue changes in the lung in

emphysema are very slow in their development, but are all to be referred to the gradual yielding of the alveolar walls as a result of increased expiratory pressure, the failure of the elastic fibres permitting of such yielding. Primarily, there is dilatation of the infundibula and alveoli. As the alveolar walls become more and more stretched under the frequently recurring pressure, they become thinner. The capillaries in the walls disappear. The thinning goes on to perforation in the centre of the alveolar wall forming a partition between two alveolar spaces, and, this hole enlarging more and more, the partition finally disappears, so that two alveolar spaces are thrown into one. This larger one may coalesce with others, forming still larger cavities, and in this way blebs the size of a hen's egg or larger may form. Generally speaking, however, the alveoli are much smaller than this. In a typical case the process is diffused throughout the lung, though for reasons previously mentioned more diffuse along upper borders and in the lingula.

Such a process means the disappearance of a large amount of the aërating surface of the lung, with extensive capillary regions, so that not only is there much less capacity on the part of the lung for performing its normal function, but the lessened vascular area causes a greater resistance to the passage of the blood through the remaining capillaries in the alveolar walls, consequently higher tension in the pulmonary artery, which in turn leads to hypertrophy of the right ventricle.

A certain amount of collateral circulation is established through the anastomoses of the branches of pulmonary and bronchial arteries, but this is relatively slight.

With the progressive diminution in the amount of elastic tissue in the alveolar walls the expiration becomes more and more prolonged and labored, and the chest assumes more and more the position of permanent inspiration.

This change in the respiratory act leads early to circulatory disturbances. Normally, in inspiration there is a negative pressure in the thorax of 7 to 9 m.m. mercury. This leads to aspiration of blood from the veins toward the heart. But in emphysema, with the lessened elasticity of the lung this negative pressure is diminished, so that relatively early in the disease distention of the veins above the clavicle and moderate cyanosis of the face are present. These appearances are striking long before the relative insufficiency of the tricuspid, due to dilatation of the right ventricle, occurs.

The anatomical changes in emphysema, already described, lead to marked changes in the expiratory pressure and in the lung capacity.

The expiratory pressure can be readily determined by a form of manometer suggested by Waldenburg and called by him a pneumatometer. In the normal individual it ranges from 110 to 130 m.m. mercury, but in emphysema it varies from 80 to 100 m.m. The inspiratory pressure remains the same as in health, 70 to 80 m.m. Measurement of the lung capacity or volume of air shows it to be about 3500 c.c. for a healthy individual, but not more than 1000 to 2000 c.c. in emphysema.

What is striking at the autopsy in a case of emphysema are—the large chest; the low position of the arch of the diaphragm; the non-retraction of the lungs on removal of the sternum; their pallor; the

overlapping of the lungs in the median line, the heart being covered by them.

The lungs are very voluminous, the lack of elastic fibres preventing their retracting. They do not crepitate, but when squeezed collapse, giving a downy feel to the hand like that of a down pillow. They are inelastic, the enlarged alveolar spaces showing beneath the pleura. To the naked eye they show a coarse, spongy structure riddled with holes. The finer changes have already received attention. Elsewhere in the body, in advanced cases, may be found the results of chronic passive congestion with dropsy, due to the obstruction to the passage of blood through the lungs.

CLINICAL MANIFESTATIONS.—Vesicular emphysema is nearly always a very chronic disease. The onset is gradual and the progress slow. Years intervene before the fatal result occurs.

The clinical picture is usually modified by coexistent bronchitis and asthma. In nearly all cases the bronchitis is the primary affection, but occasionally the emphysema precedes the bronchitis, the circulatory disturbances resulting from the emphysema favoring attacks of bronchitis.

In all cases of substantive emphysema, excluding hereby the senile emphysema and Louis' cases occurring in children, there is a close relation between the bronchitis and the emphysema.

The symptoms resulting from emphysema are directly dependent upon the tissue changes described—diminution in aërating surface, loss of capillaries, lack of elastic tissue. These changes naturally lead to dyspnœa and difficulty in expiring the air. The term expiratory dyspnœa best expresses the condition.

In the early stages the dyspnœa is felt only on exertion, which is much increased in bad weather by the tendency to exacerbations of bronchitis. Paroxysms of cough or asthmatic attacks add greatly to the patient's distress in breathing. The expectoration is usually very slight, what there is being tenacious mucus.

As the disease advances the dyspnœa becomes more urgent, the expiratory effort increases, and, to add to the discomfort, the right ventricle, which had previously hypertrophied to compensate for the reduced vascular area of the lung and the higher resulting pressure in the pulmonary artery, dilates; relative insufficiency of the tricuspid takes place, and with it the resulting venous engorgement of the organs, with anasarca and dropsy of the serous cavities. In other words, the picture is much that of an advanced mitral lesion with broken compensation.

Cyanosis is usually present before insufficiency of the tricuspid occurs, for reasons previously given. After the tricuspid becomes insufficient the cyanosis is intensified and permanent, and still further increased during cough.

Cough is present in most cases.

The forcible character of the expiration is often apparent to the ear, and, when the element of asthma is added, becomes loudly wheezing.

PHYSICAL EXAMINATION.—*Inspection.*—The anatomical changes in the lung lead to alterations in the shape of the chest that are very characteristic. The chest assumes the position of permanent inspiration, the suggestion to the observer being that the patient has taken an un-

usually full breath and is holding it. In time the chest, as a whole, becomes barrel-shaped. The antero-posterior diameter is increased so as to equal the transverse diameter, whereas normally it is only three fourths that diameter. The sternum is arched forward, the cartilaginous ribs are prominent, and the back in the dorsal region is hollowed. The sterno-cleido-mastoidei and the scaleni, aiding as they do in the respiratory effort, hypertrophy and stand out prominently. The supraclavicular region is full and the neck short. The thorax is relatively shortened, the intercostal spaces are wide, and the ribs move but little. Along the lower border of the ribs enlarged veins may be seen in many cases. The veins of the neck are prominent, and, after tricuspid leakage follows, pulsate.

Inspiration is short and jerky. Expiration is prolonged and labored, and the aid given to it by the abdominal muscles is visible to the eye. The apex beat of the heart is indistinct; when seen it may be in the sixth or seventh interspace, not from hypertrophy of the left ventricle, but because the heart has been pushed down by the enlarged lung. Pulsation in the epigastrium is common, being the impulse of the hypertrophied and depressed right ventricle.

The above series of changes, to be observed by the eye alone, are so characteristic as to often make the diagnosis of emphysema certain. When the tricuspid becomes insufficient there is added general dropsy.

Percussion.—The resonance is occasionally vesiculo-tympanitic or tympanitic; more commonly hollow or drumlike. The area of cardiac dulness is diminished in all cases, owing to the fact that the hypertrophied lung covers the heart to a greater or less extent. In extreme cases no cardiac dulness is to be obtained. Generally speaking, the cardiac dulness begins at the fifth or sixth rib, the heart being pushed down as well as partly covered by the emphysematous lung.

The lower line of pulmonary resonance may be two or three ribs below the normal, and, as the liver is also lowered with the depressed diaphragm, its inferior border is proportionately lower, its edge being easily palpated.

The supervening hydrothorax and ascites are readily made out by percussion.

Auscultation.—The respiratory murmur is faint both in inspiration and expiration, the notable characteristic being the prolongation of the expiratory sound, which is usually weak.

Owing to the frequency of bronchitis, sibilant and sonorous râles, with occasionally moist râles, are to be heard.

When associated with asthma the respiratory murmur becomes distinctly wheezing.

The apex sounds of the heart are diminished in intensity. The systolic apex murmur often heard is functional and not organic. The second pulmonic sound is accentuated, owing to the compensatory hypertrophy of the right ventricle. The right ventricle sounds are distinctly heard in the epigastrium. A tricuspid systolic murmur is to be heard when the tricuspid valve becomes insufficient.

The enlargement of the liver and spleen in the later stages can usually be made out. The urine in this stage is characteristic of the passive congestion of the kidneys.

COURSE; PROGNOSIS.—The prognosis of the disease depends upon the station in life of the patient and the climate. Those in the well-to-do class, who are neither exposed to inclement weather nor obliged to do physical work, live many years in comparative comfort. In summer they can be out of doors without risk of bronchitis or of increasing the bronchitis already present, while in winter they can remain housed or go to a warmer climate. In the changeable weather of spring and autumn there is risk of bronchitis, and so an increase of the trouble.

Among the poorer classes, with the necessity of manual labor and exposure to all sorts of weather, emphysema gives more discomfort, progresses faster, and leads sooner to failure of cardiac compensation.

In all uncomplicated cases the duration is to be measured by years. Patients with emphysema often die of an intercurrent disease, such a disease being in itself fatal or else proving more dangerous than it would in an otherwise healthy person, owing to the weakness of the right ventricle of the heart.

If no intercurrent disease occurs, death results from the general dropsical condition and the nutritive disturbances associated with chronic passive congestion of the organs.

TREATMENT.—The nature of the tissue changes in the lung in emphysema prevents any hope of a cure being entertained. What can be done is to lessen the tendency to bronchitis and to help that condition when present. Change to a warmer climate in winter is an advantage. For those who cannot make this change, but who are not compelled to work, indoor life in winter is preferable. Avoidance of over-exertion, where possible, is desirable.

When one views the matter of treatment from the changed characters of the respiratory act already explained, it becomes obvious that if the patient could inspire compressed air and expire into rarefied air, much relief would be afforded. An apparatus for accomplishing this purpose has been devised by Geigel-Mair, but it is costly, hence within the means of but few. Moreover, its use hardly does more than give temporary relief.

As medicinal means iodide of potash, citrate of potash, and pilocarpine are useful for the bronchitis. Strychnine is of value in all cases of the disease.

Nutritious diet and care of the bowels, by improving the general condition, help the patient.

When compensation of the right ventricle fails, with the results attendant upon the general venous engorgement, the treatment is like that of an uncompensated mitral lesion—namely, rest, digitalis or strophanthus, strychnine, calomel for its diuretic action, or diuretin.

SENILE EMPHYSEMA.

Emphysema of the aged is simply part and parcel of the general senile atrophic changes. The disturbance in the function of the lung is usually not out of proportion to that of other organs; consequently, these are not predominant respiratory symptoms.

In senile emphysema the chest is usually somewhat flattened; the chest hyper-resonant; respiratory murmur faint, and expiration pro-

longed. The condition does not give rise to increased pressure in the pulmonary artery, hence does not call for more work on the part of the right ventricle.

At the autopsy it is found that the lungs are not voluminous; they do not cover the heart; they do not retract on removal of the sternum; are pale; do not crepitate; and on squeezing can be reduced to a very small volume.

Beyond slight cough and some shortness of breath there are no symptoms.

As to the TREATMENT, there is nothing special to be done.

ATELECTASIS.

DEFINITION.—By the term "atelectasis" it is meant that the lung or a portion of the lung is not distended with air—that is, that the alveoli of the affected part contain no air.

From its derivation the word means incompletely distended, and was originally applied to the condition often seen in the newborn, where part of the lung is distended with air, the rest being in a state of collapse.

ETIOLOGY.—As is well known, the lungs of the child before birth and of the stillborn contain no air. This is complete fœtal atelectasis.

In weakly children after birth, and in those prematurely born, the lungs frequently do not fill with air throughout. The air does not enter certain portions, probably from weakness of respiratory effort, and such portions remain in the collapsed state. A similar result is sometimes seen in the newborn from inhalation of meconium or mucus, thus plugging bronchi and preventing the entrance of air. This is partial atelectasis of the newborn. It is often recovered from, but opportunities for seeing the condition anatomically are frequent, as such children are apt to die early. The contrast to the eye between the pink inflated portions of the lung and the dark red, sunken, airless portions is very marked.

Formerly this partial fœtal atelectasis was considered a form of pneumonia, but Jörg in 1832 showed clearly that the condition was simply one of persistent collapse of alveoli due to non-entrance of air. Artificial inflation of the collapsed portions is so readily performed as to demonstrate at once that a pneumonic process is not present.

Opposed to fœtal atelectasis, in the classification of the condition, is the acquired form. A portion of a lung or a whole lung may be deprived of its air in one of two ways—either by the plugging of a bronchus, large or small, or by compression of the lung from without.

It is a well-known fact that if a bronchus or bronchiole becomes plugged by secretion or by a foreign body, the air in the lung beyond the occluded bronchus is absorbed by the blood. Hence collapse of the alveoli supplied by that bronchus takes place.

This form of atelectasis is one of the important steps in the series of changes occurring in broncho-pneumonia, in which, as is well known,

bronchitis, plugging, atelectasis, œdema, exudation, and consolidation represent the consecutive changes.

In the capillary bronchitis of young children and old people atelectasis is likely to occur.

Partial atelectasis is readily recognized anatomically by the fact that the portions involved are dark red in color and lie below the level of the surrounding normal lung. This appearance is best seen on the pleural surface.

In those ill in bed in the dorsal decubitus, with severe acute febrile disease or in chronic marantic conditions, atelectasis of the dependent parts is apt to occur, and to be followed by œdema. This is caused by strain upon the weakened circulation.

The term atelectasis is also used in a less strict sense to include absence of air due to compression of the lung by causes acting from without, such as pleuritic effusions, hydro-thorax, pneumo-thorax, and tumors.

The effect upon the lung of moderate effusions is not to produce atelectasis, but a retraction of the lung occurs in virtue of the elasticity of the alveolar walls. It is only in extensive effusions that the air is wholly expelled from the lung. The term carnification is more commonly used to express this form of atelectasis.

Atelectasis of a considerable degree may be present in the lungs of individuals with kypho-scoliosis. In this condition bronchitis is apt to occur, and is peculiarly liable, from the conformation of the chest, to be followed by atelectasis.

Symptoms.—When atelectasis appears by itself it is often possible to diagnosticate it clinically. Far oftener it is so intimately connected with the associated processes already named that in diagnosis and prognosis it cannot be separated from them.

In its pure form atelectasis is not associated with fever. A considerable degree of atelectasis may exist without modification of the shape of the chest or influencing the position of neighboring organs. There are, however, changes in the character of the respiration and in the area of cardiac pulsation and dulness. The respiration is increased in frequency according to the extent of lung involved. During inspiration the soft parts above the clavicle and the intercostal spaces and the lower parts sink in, due partly to increased contractions of the diaphragm, partly to atmospheric pressure.

Cardiac dulness is increased, because the lung is smaller; consequently, more of the heart is uncovered. Percussion of the lung gives dulness or flatness according to the extent of the process. The respiratory murmur is diminished unless a large area is involved, when it may be bronchial.

Treatment.—As to treatment, prevention of the causes is the only means within reach.

ŒDEMA OF THE LUNG.

Definition.—Œdema means a collection of fluid in the alveoli and smaller bronchi. The fluid comes from the blood, being a result of transudation through the vessel wall. It is usually clear, but may be tinged red from the presence of red blood corpuscles.

Etiology.—The ultimate cause is always a circulatory one, including under this general heading changes in the character of the blood, local changes in the vessel wall, and variation in the blood pressure. It is a common condition in diseases of the heart associated with passive congestion, in chronic renal disease, and in inflammatory processes in the lung.

In cardiac disease the variation in blood pressure accounts for it. In renal disease associated with hydræmia there are doubtless changes in the vessel walls, whereas in chronic interstitial nephritis the weakening of the heart in the late stages is a more probable cause. In the neighborhood of inflammatory foci in the lung œdema is likely to occur; hence this has been called inflammatory, and doubtless represents an actual inflammatory exudation.

The importance of œdema is variable. Occasionally it is important, and may be the cause of death. In the majority of instances, however, it is not to be considered the cause of death, but rather a result of the disease that is proving fatal. Cohnheim has well stated the case by saying that an individual does not die because he has œdema of the lung, but that he has œdema of the lung because he is dying. By this it should not be inferred that œdema occurs only when a person is in a dying condition, for it is often transitory, appearing and disappearing many times in the course of chronic cardiac or renal disease.

Welch's experiments show that many cases of œdema are due to congestion which results when the left ventricle is paralyzed, while the right ventricle continues to force the blood into the lung.

In the majority of cases the importance of the œdema is overshadowed by the disease that has led to the œdema.

Pathological Anatomy.—The process may be limited or diffuse. When limited it may be present in the tissues surrounding an area of inflammation, or it may, from gravity, manifest itself in the dependent parts of the lung. The diffuse form usually begins at the base, extending with greater or less rapidity to the upper parts of the lung.

Anatomically, the condition is readily recognized. The lung is more voluminous, does not retract so completely as normal, nor does it crepitate as well; the density is increased. On section it shows a moist surface, and on squeezing yields considerable thin aërated fluid.

Symptoms.—Clinically, the symptoms of œdema are due to the mechanical effects of the fluid in the alveoli. But it should be borne in mind that in the majority of cases in which signs of œdema are found the symptoms are much more dependent upon the original disease than upon the œdema. Dyspnœa, cyanosis, labored respiration, with increased frequency, are the important symptoms. Physical examination shows in case of œdema, such as is apt to occur in the course of an uncomplicated mitral lesion or in nephritis, fine moist râles in the lower

part of the lungs posteriorly, without change in the percussion resonance. As the œdema increases the râles are heard higher up in the chest, and the fluid in the lower portions, collecting in greater quantity, expels much of the air. The lung thus assumes more and more the characteristics of a solidified lung, giving dulness or flatness on percussion, and often broncho-vesicular or bronchial respiratory murmur.

The onset of œdema may be rapid or gradual. Frequently it reaches a certain degree and then retrogrades, depending upon the strength of the circulation. It is liable to come on suddenly in the course of the diseases already mentioned, and is associated with great dyspnœa and cough, with the expectoration of a foamy matter resembling white of egg beaten up. In many cases such a foam pours out of the mouth.

Closely allied to the above in symptoms is a form of acute general œdema of the lung coming on suddenly and often proving fatal in an hour or two. At the autopsy nothing may be found beyond the appearances characteristic of œdema of the lung, but it is fair to assume that it results from cardiac weakness.

Treatment.—As to treatment, it may be said that, inasmuch as the œdema is almost always dependent upon weakened heart action, the effort should be made to strengthen this organ by vigorous hypodermic stimulation.

In the severe cases, with engorgement of the lung, bleeding is proper.

In the very acute cases, without apparent heart or kidney trouble, described above, a hypodermic of morphine is sometimes of help. Cupping the chest is of great service, and the inhalation of oxygen gas sometimes gives relief.

ABSCESS OF THE LUNG.

Suppurative processes in the lung occur under very varying circumstances, and are often so closely connected with other inflammatory and necrotic changes as to form merely a part of a combination. A simple suppurative process is uncommon. Nearly all the conditions to which the name of abscess is given represent a combination of more or less circumscribed pneumonias, necroses, suppurations, and putrefactive changes.

Etiology.—Abscesses may be single or multiple. They may represent the terminal stage of some local process in the lung, like fibrinous or broncho-pneumonia, or they may result from an extension of suppuration from a neighboring part, as in empyema, mediastinitis, perforation of cancer of œsophagus; or they may represent the advance through the lung of pus from an abscess of the liver on its way to discharge through a bronchus; or, finally, the abscesses may be of embolic origin.

As stated, simple suppuration in lung tissue rarely occurs; for, owing to the pressure of air in the lung, with the micro-organisms present such as usually occur in air, necrotic and putrefactive changes are likely to be associated.

Abscess occurring in the later stages of acute fibrinous pneumonia is usually single and large, but when abscesses are the result of bronchopneumonia, especially the inhalation variety, they are multiple and scattered, because the foci of inflammation are multiple. Both of these forms of abscess have received attention under the headings above given. (See pages 202, 204.)

Foreign bodies almost invariably lead to suppuration in their neighborhood, and thus an abscess is formed. Owing to associated putrefactive changes the pus is dark-colored, foul-smelling, with shreds of lung tissue in it.

Empyema bursting into the lung with discharge of pus by the mouth occasionally occurs, and the pus in its passage through the lung may rarely cause suppuration in the lung itself. The fact of the perforation is at once apparent from the pus coughed up, but the question whether the lung is also involved through infection by pus-producing micrococci, or whether the lung and bronchi simply furnish an outlet for the pus, is difficult to decide. The existence of the empyema itself interferes greatly with the physical examination of the lung of the affected side.

Sub-diaphragmatic empyemas, without the signs of an ordinary empyema, may in the same way open into the lung with expectoration of pus.

Abscess of the liver, especially the large solitary variety secondary to amœbic dysentery, as well as the abscess resulting from suppuration in an echinococcus cyst, may rupture into the lung, adhesive inflammation between liver and diaphragm and diaphragm and lung having previously occurred. In the pus thus expectorated bile pigments, bits of the necrosed liver substance, together with amœbæ coli or echinococcus hooklets, according to the character of the abscess, are frequently found, and are a valuable aid in diagnosis.

Multiple abscesses due to septic emboli are of frequent occurrence. Such septic emboli are derived from thrombi in a state of septic softening. The location of these thrombi may be as follows: In the lateral sinus of the dura mater secondary to a phlebitis, due in turn to an extension of suppuration from the middle ear. This is of considerable clinical importance, for not a few of the fatal cases of middle-ear suppuration owe their termination to multiple abscesses in the lung. The septic thrombi may be situated in the uterine sinuses and veins, the result of diptheritic endometritis following childbirth or abortions or any lacerations of the interior of the uterus, associated with septic infection; or the thrombi may be situated in the inferior hemorrhoidal plexus. Occasionally, patients operated upon for piles die of abscesses of lung from septic softening of the thrombi of the ligated veins, with transfer of septic emboli to the lung. Thrombi in the pelvic plexuses or in wounds on the external surface of the body, the veins of which empty into the cava, may, after infection and septic softening, furnish septic emboli.

Such embolic abscesses of the lung are multiple, usually small, averaging the size of a pea. Though scattered through the lung, they are always more numerous beneath the pleura. This situation is a matter of importance, for as long as the patient lives these abscesses tend to extend peripherally. In this way the pleura is sooner or later involved; circumscribed necrosis of the pleura with perforation takes

place, with discharge of contents of the abscess into the pleural cavity, leading to a severe and rapidly fatal form of septic pleurisy.

Diagnosis.—This form of abscess can be diagnosticated if one bears in mind the anatomical relations between the primary disease, be it in the ear, uterus, or wounded surface, and the likelihood of the results of septic embolism, which under these conditions can lodge only in the lung. Given the source of embolism and symptoms of pain in the side, chills, irregular fever, quickened and shallow respiration, the chances are in favor of abscesses in the lung. If a friction sound is heard, perforation has occurred or is about to occur. In some cases of thrombosis in the lateral sinus the thrombus may extend downward into the jugular vein in the neck, and thus be felt on palpation. This, of course, adds greatly to the probability of abscess formation in the lung.

As to the treatment of these cases of multiple septic abscesses, it may be said to depend upon care of the original cause, with constitutional treatment for keeping up the strength.

The larger, generally solitary, abscess of the lung usually discharges through the mouth, in which case the diagnosis at once becomes easy, or perforation into the pleural cavity, causing pyopneumo-thorax, may result. Occasionally an abscess situated near the pleura causes adhesion of the two surfaces; hence perforation with discharge into the pleural cavity is impossible. Such a case occurred in the service of the writer at the Massachusetts General Hospital. Clinically, it was thought to be a circumscribed empyema. Aspiration gave pus, and the case was then transferred to the surgeon for permanent opening and drainage. The operation showed the case to be one of abscess of the lung directly under the costal wall, the pleural cavity having been shut off by adhesions. Recovery took place.

Unless there has been an expectoration of pus there are scarcely any signs that enable one to diagnosticate the larger abscess of the lung. Occasionally one reaches a diagnosis by eliminating other possibilities. The means of inferring its existence when secondary to pneumonia have been considered under that heading (page 205). Persistent leucocytosis is also a help.

Prognosis.—The prognosis of a simple abscess is often favorable, recovery taking place by discharge of the contents and healing and retraction of the walls. When the abscesses are multiple and due to septic emboli the outlook is very unfavorable.

Treatment.—As to treatment, it must be expectant in the case of larger abscesses, unless they are situated near the chest wall; in which case opening with drainage should be tried. If it can be shown with a tolerable degree of certainty that an abscess is present in a portion of lung within reach of the knife, it is hardly justifiable to treat the case expectantly, owing to the danger of perforation and discharge of pus in some other direction than through a bronchus.

When a patient is expectorating a purulent fluid it is of importance to determine whether the material comes from a bronchus or bronchiectatic cavity on the one hand, or from an abscess on the other hand. Both may be, and usually are, bad smelling, but on microscopic examination of the pus from an abscess one usually finds bits of lung tissue or at least elastic fibres.

GANGRENE OF THE LUNG.

Definition.—By the term gangrene is meant a putrefactive process occurring in dead tissue. For its occurrence in the lung it is necessary that a portion of the lung necrose, and that bacteria of putrefaction gain access to this dead part. These latter may enter the lung directly or in bits of organic matter, such as food, inhaled into the air passages.

Etiology.—Many of the conditions that lead to abscess lead also to gangrene, and under the heading Abscess of the Lungs will be found a consideration of them (page 238).

Any inflammatory process in bronchi or lung tissue may be associated with a putrefactive change in the product, and thus gangrene arises. The necrosed portion of lung tissue resulting from cutting off the blood supply or from the direct action of septic bacteria may become putrid—that is, gangrenous.

In lobar pneumonia; in broncho-pneumonia, especially the inhalation variety; by perforation into the lung of new growths or abscesses; in infarctions from bland or septic emboli,—gangrene may supervene, either in single larger area in lobar pneumonia, or in smaller, usually multiple, areas in other conditions. Gangrene is more common in the lower lobe.

Pathological Anatomy.—Anatomically, areas of gangrene present a characteristic appearance. The lung tissue is much softened, almost diffluent, of a dirty grayish black color. If no actual excavation has taken place, there may at first sight appear to be relatively little change in the lung tissue, but if a stream of water is allowed to flow over the cut surface, it will be found that little of lung is left except a fibrous network. If the individual lives longer or if connection with a bronchus takes place early, then a cavity exists with ragged, irregular, shreddy walls. The odor of the lung, in whatever stage the individual may have died, is very foul and of an exceedingly penetrating character.

Around the area of gangrene may always be found an inflammatory process, either a reactive one or else the remains of the product present previous to the onset of the gangrene.

Symptoms.—Clinically, the symptoms and course of gangrene will depend much upon the character of the process preceding the gangrene. Usually the first indication that gangrene has supervened is given by the character of the expectoration and the odor of the breath. The sputum is abundant, rather thin, foul-smelling. If collected in a glass and allowed to settle, it shows an upper layer made up of muco-purulent material, mostly in the form of balls; a middle layer, thin and watery; and a bottom layer, mostly purulent, with greenish shreds in it. Microscopically, there are to be found shreds of lung tissue, notably elastic fibres; also fat crystals, free fat, detritus, and bacteria in enormous quantities. The sputa in putrid bronchitis show no elastic fibres. The odor of the breath is persistently foul—not that of ordinary putrefaction, but peculiarly stinking and penetrating. In fact, it is often possible to make a diagnosis of gangrene of the lung on entering the sick-room.

The odor of the breath calling attention to the existence of gangrene

somewhere in the lung, it may be possible to localize it if of considerable area and near the surface. Dulness on percussion and bronchial respiration are likely to be present. If it has gone on to excavation, the signs of a cavity may be found, cracked-pot modification of tympanitic resonance, and amphoric respiration.

Fever varies much in different cases both according to the pre-existing inflammation in the lung, its distribution, and also whether there is free drainage into the bronchus with expectoration of the putrid material. In the latter case the fever is less than in the former.

CLINICAL COURSE.—The course depends much upon the cause, as explained under the section Abscess of the Lungs, as does also the prognosis. The disease may last days, weeks, or months, and may end in recovery, but this outcome is the exception rather than the rule, the patient usually dying of exhaustion, though hemorrhage from erosion of a vessel, and rarely abscess of the brain, may be fatal.

TREATMENT.—The treatment of gangrene is largely expectant. Measures to keep up the general strength occupy the first place. The odor can be much lessened and the patient given some ease by inhalation of equal parts of beech creasote, spirits of chloroform, and alcohol in a Robinson inhaler.

If near the surface of the lung, surgical interference for drainage should be thought of, but the results thus far have not been promising.

A consideration of gangrene as a complication of pneumonia will be found upon pages 238 and 240.

PULMONARY EMBOLISM.

PATHOLOGY.—Embolic processes in the lung vary greatly in the anatomical changes and in the clinical manifestations according to the character of the emboli. The emboli lodging in larger or smaller pulmonary vessels may be solid, liquid, or gaseous.

Solid emboli are usually portions of thrombi which may be bland or septic in character. The liquid emboli are fat in a fluid state. The gaseous emboli are bubbles of air introduced into the circulation through open veins in which there is negative pressure, as in the neck and uterus under certain conditions.

The solid emboli are derived from thrombi in the right auricle or auricular appendage, in the right ventricle, in the cava and its tributaries, especially the venous plexuses of the pelvis, and the veins of the lower extremities. If the thrombus from which the emboli are detached is in a state of septic softening, due to the presence and action of pathogenic bacteria, then the emboli will be possessed of similar infective characteristics, and will cause necrosis and inflammation in the tissues around the vessels in the lung in which they lodge. The results of this form of septic embolism have been described under the heading Abscess of the Lungs (page 238).

If the emboli are bland, they exert, primarily at least, only a mechanical effect, this effect varying according to the size of the vessel plugged.

An embolus may be large enough to block the primary pulmonary artery. Such an embolus usually comes from the inferior cava, the result of an extension into it of a thrombosis of the pelvic plexuses or of the veins of one of the legs. A thrombus starting in a leg or in a venous plexus of the pelvis tends to extend by further clotting toward the heart. After reaching and extending into the inferior cava, it is apt to be broken off by the current of blood entering the cava from the common iliac vein of the opposite side. Carried upward with the blood current, it passes through the right side of the heart, and, if too large to pass into the right or left pulmonary artery, straddles the bifurcation and greatly hinders, and frequently wholly prevents, the passage of blood through the lung. This is by no means of uncommon occurrence, and the symptoms associated are of the most pronounced character.

Symptoms.—Suddenly, without any warning, the patient feels a shock in the chest, followed by great distress in breathing, restlessness, and anxiety. The sensation of suffocation is intense, and the patient makes powerful respiratory movements in the hope of getting more air. More air he readily gets, for there is no obstruction to its entrance into the air passages, but, as the circulation of blood in the lung has come to a standstill, there is of course no aëration, and the patient dies in a few moments cyanosed. The onset is sudden, the suffering terrible, the end rapid. Clinically, embolism is rarely definitely anticipated, though often thought of when a source is present in the thrombosis of veins of the leg. Puerperal women occasionally die of it, the thrombosis in this case having its origin in the uterine sinuses or pampiniform plexus. Occasionally in the course of the acute infective diseases a thrombosis starts in the pelvic plexuses and extends toward the heart, with the results already described.

Sometimes the embolus is of such a size as to enter and block one pulmonary artery, the other remaining pervious to blood. In this case the symptoms are like those mentioned previously, but less urgent. The result, however, is none the less fatal, though delayed, sometimes, for hours. The explanation is as follows: The lung whose pulmonary artery is plugged receives no blood, and, though well supplied with air, cannot aërate the blood, as it does not circulate in it. The other lung receives double the amount of blood, which leads to œdema; consequently, the air does not enter that lung sufficiently and the individual dies of suffocation.

When the emboli are smaller they naturally find their way into smaller arteries and plug them. As the lung has terminal arteries—that is, arteries without arterial anastomoses—the portion of lung supplied by the occluded artery undergoes necrosis, with back flow of blood into the part from anastomosing capillaries, which fills it with blood, forming the characteristic dark red hemorrhagic infarction.

The sources of the emboli have been mentioned above, but in the production of the hemorrhagic infarction the right side of the heart is the usual origin, the circulation through it having been weakened as a result of mitral lesions. In other words, when the compensation of a mitral lesion fails, thrombosis of the dilated right auricle and ventricle is common.

Hemorrhagic infarctions the result of bland emboli may remain bland

and undergo absorption as a result of the secondary, reparative inflammatory process set up about them; but in many cases, owing to the fact that this dead mass is exposed to the air in the lung, secondary putrefactive or infective or suppurative processes may be set up in it as a result of the action of bacteria brought to it in the inspired air. In this way abscesses or circumscribed putrefactive processes (ordinarily called gangrene) may arise.

Clinically, embolic infarctions may or may not give rise to symptoms. They are frequently found at the autopsy without there having been any indication of them during life. If situated in the interior of the lung, they rarely cause more than a temporary expectoration of bloody material. If beneath the pleura, they often cause sudden localized shock at a point of lodgement of the embolus, and later a stitch in the side due to the secondary local pleurisy.

Fat embolism of the lung is a condition seen after injuries associated with extensive crushing of fat tissue and of the marrow of bones. Crushing wounds of the thighs, such as result from a person being run over, lead to the setting free of considerable fluid fat. This is taken up by the lymphatics and veins. The veins deliver it directly to the lung, the lymphatics indirectly through the thoracic duct, opening as it does into the left subclavian vein. The oil drops, not moistening the lining of the capillaries, cannot pass them, and so block the passage of blood. If the amount of fat set free is small, the amount of plugging of pulmonary capillaries leads to no injury to the individual. If the amount is large, death results under symptoms of suffocation.

The usual history is that on the day after, sometimes on the second day after, the receipt of an extensive crushing injury the patient begins to show a dusky color and greater frequency of respiration. These symptoms increase greatly in the course of the next twenty-four or forty-eight hours, with death under signs of incomplete oxygenation of blood.

At the autopsy the lungs show little or nothing, so far as gross appearances go, but if a bit be snipped off with scissors and examined under the microscope, there will be found great numbers of glistening oil drops, often fused into various shapes, corresponding to the contour and divisions of the capillaries.

The reason of the late onset of symptoms and relatively slow progress is due the fact that the liquid fat is slowly absorbed at the wound. This can be recognized clinically if the facts mentioned above are borne in mind in connection with such injuries. Naturally, nothing can be done in the way of treatment of the embolic process.

Air Embolism.—In air embolism a part of the air may be found in the lung capillaries, but most of it remains in the right side of the heart. Air may enter the veins during surgical operations about the neck from accidental opening of a vein. The pressure being negative during inspiration, air enters rather than blood escapes. In the uterus after confinement, especially if for any purpose the knee-elbow position be assumed, air may enter through the vagina, and then make its way into the uterine sinuses, which in this position may have a negative or nearly negative internal pressure. At any rate, with the resumption by the patient of the dorsal decubitus whatever air had previously entered the

uterus would be subjected to greater pressure, and in this way be forced into the veins. Frequently in the attempts of irregulars to bring about abortion, air and water, through a leaky syringe, are forced between the membranes, which are forcibly separated, the sinuses opened, and air carried into the sinuses. This air, carried to the right side of the heart and to a less extent to the lungs, gives rise to intensely sudden dyspnœa and death in a moment or two.

PNEUMONOKONIOSIS.

DEFINITION.—The konioses are diseases resulting from the inhalation of dust, the mechanical and not, at least in the early stage, the bacterial effects being manifested.

ETIOLOGY.—Dust of varying character is inhaled by all persons—to a lesser degree by those living in the country, to a greater degree by those in town. Nearly all this inhaled dust is got rid of by the action of the cilia of cells lining the air passages and cough. The little that remains in the lung is practically of no importance.

The question of the introduction of dust becomes of interest when it is inhaled in large quantities, as in certain occupations, especially so when the particles themselves are sharp, so as be more readily taken into the lung tissue. It is not to be inferred by this last statement that particles usually make their way into the lung tissue in virtue of their sharp edges; on the contrary, most of the particles that find their way into the lung itself are taken up by epithelial cells or leucocytes, and carried bodily into the lymphatics in the walls of the finer bronchi or alveoli. Some remain here; others are carried on in the lymphatics until they reach the bronchial lymph glands, where they remain permanently.

It is well known that the connective tissue stroma forming the interlobular septa is rich in lymphatics. These are especially a place of deposit for inhaled particles, which by their color, whatever it may be, serves to mark out, anatomically, very beautifully the lobular regions, especially beneath the pleura.

Some of the inhaled particles enter the walls of the finer air passages, and pass directly into the lymph spaces without the intervention of wandering cells possessed of amœboid movements.

Although most of the particles that enter the lymph spaces are retained in them or in the sieves of the lymph current, the bronchial glands, yet some pass through the lymph glands and make their way into the general circulation, and in turn find their way into those well-known repositories of pigment, the liver and spleen.

Even in those who inhale large quantities of the varieties of dust to be presently described but little, relatively, is permanently stored up in the lung tissues or contiguous lymph glands. Most of it is got rid of by expectoration.

The part played by the presence of the gritty particles is two fold—that upon the bronchial mucosa and that upon the tissues forming the

stroma of the lung. The latter is the more important and will be considered first.

Pathological Anatomy.—In appreciating the character of the inflammation set up by these particles it is desirable to bear in mind the fact that the lymphatics lie in the connective tissue; hence, as the particles lie in the lymphatics, any irritation they might cause would manifest itself by changes in the surrounding connective tissue. This is found to be constantly the case. Proliferation of the connective tissue, with the production, at first, of a more cellular, later, a more fibrous, tissue, is the result. Such thickening of the alveolar walls leads to compression of the vessels in them, also encroachment upon the alveolar spaces. The thicker the walls become the smaller the alveolar spaces become, so that in time the portion of lung especially affected may be impervious to air. This represents a typical chronic interstitial pneumonia. Thickening of the peribronchial connective tissue is very common.

In still later stages a softening in this new-formed connective tissue may occur with formation of cavities. This is undoubtedly a necrosis, and is probably closely connected with the presence and action of tubercle bacilli, which, although not present in the early stages of the affection, are likely to be later. Hence a tubercular process is engrafted upon a chronic interstitial pneumonia.

The relation of the tubercle bacillus—that is, tuberculosis—to this form of interstitial pneumonia is one of great interest. In some cases tubercle bacilli are found in the sputa, in other cases not. Much more study and observation is necessary before satisfactory conclusions upon this subject can be drawn. When a tubercular process supervenes, it is not to be supposed that it is a direct result of the inhalation of the dust, but rather that changes produced in the lung by the dust furnish a satisfactory soil for the growth of tubercle bacilli.

The anatomical processes described nearly always affect the apices of the lung to a far greater degree than the remaining portions, for the reason that the apices are less well "ventilated;" that is, the air currents are, in ordinary respiration, feebler there than elsewhere in the lung. Hence the gritty particles are less well disposed of, and are retained in those portions to a much greater degree.

The second effect due to the presence of gritty particles is bronchitis. This is nearly always present, associated with cough and expectoration of mucous or muco-purulent fluid, stained the color of the inhaled material when that has a color, and in which, microscopically, may be seen the particles of dust. This bronchitis is chronic, and in its course is likely to lead to emphysema with its characteristic dyspnœa. (See Emphysema, page 229.)

Varieties of Dust Inhaled.—The kinds of dust inhaled are as numerous as the trades involving inhalation of gritty particles. The more important of them only will receive attention.

The three common forms of pneumonokoniosis are those due to inhalation of particles of coal, iron, and minerals used for grinding purposes.

(1) *Anthracosis.*—The disease due to inhalation of particles of coal is called anthracosis pulmonum or anthraco-pneumonokoniosis, the latter

being the better term, though rather unwieldy. Generally, the term anthracosis, or coal-miner's lung or coal-miner's phthisis, is used.

Coal-miners and heavers inhale enormous quantities of finely divided coal. By far the larger quantity is expectorated, as is obvious by the color of the sputa. The particles are not as sharp as are those of iron and stone, and their absorption by the lung is much more diffuse; that is, a miner's lung is likely to be diffusely black. Moreover, it may be very black, showing previous absorption of a large amount of coal, without evidence of much structural change in the lung tissue. In other cases the changes described earlier in this article may be present.

(2) *Siderosis.*—Under the heading siderosis is included the results in the lung tissue due to inhalation of sharp particles of metal, especially steel. This is noticeably the case in knife-grinders and in those who inhale the air charged with the small fragments of steel or iron abraded from a surface that is being cut or ground or polished by a rapidly revolving wheel made of some mineral substance harder than the steel. Any one who has watched this process and observed the shower of sparks and the thick layer of dust covering surrounding objects will realize how much of this fine dust must be inhaled by the workman. In some shops provision is made for removing this dust by downward air currents through suitable channels, but this life-saving device is usually conspicuous by its absence.

These particles are unusually sharp and penetrate lung tissue with ease. The interstitial pneumonia caused by them is usually situated at the apex, and is very frequently followed by a tubercular process. Knife-grinder's phthisis, as it is frequently called, causes the death of many of the grinders in the cutlery shops throughout the world, notably in the great establishments in Sheffield. In conversation with those familiar with such works the writer has been informed that six years is about the limit of life.

In the grinder's trade just mentioned particles of the stone used are also inhaled, but the wear of the stone is far less than that of the metal, and proportionately fewer particles are inhaled.

The lungs in the knife-grinder's phthisis are red from the oxide of iron formed in the tissue, with predominant apex changes usually associated with tuberculosis.

(3) *Chalicosis* is the term used to express the condition arising when the particles inhaled are mineral. Any occupation involving the chipping of minerals, such as making millstones, or the abrasion of stones when used in grinding, or the frequent handling of finely-divided mineral substances, may be followed by the changes in the lung already described. Usually the lungs are pale, owing to lack of color in the particles inhaled; but sometimes they are brown from the presence of altered blood pigment, due to the hemorrhages resulting from the pricking of the sharp particles.

(4) *Miller's phthisis* is the name given to the pneumonokoniosis common in those who grind cereals. In this case the hull of the grain is probably the irritating material.

It must not be supposed that symptoms come on at once when a person engages in any of the occupations involving inhalation of gritty particles. Many months, and often years, pass before symptoms

referable to bronchitis, emphysema, interstitial pneumonia, or phthisis make their appearance.

DIAGNOSIS.—The diagnosis is to be made by taking into consideration the occupation, the sputa, the failing health, and the associated symptoms and signs of the diseases just enumerated.

PROGNOSIS.—The prognosis depends upon the stage of the disease and whether the patient can change his work. Of course, if the disease has reached such a stage that the individual gives up the dusty work, it means that it has progressed too far to be relieved.

TREATMENT.—Treatment involves prevention and cure—prevention by advising the person to use means to arrest the dust, as by inhalers, and possibly, through public health officers, the enforcement of means in grinding establishments for the removal of dust, which is entirely feasible and inexpensive.

Treatment of the disease when once established resolves itself into removal of the cause and treatment of the special indication above given.

SYPHILIS OF THE LUNG; NEW GROWTHS OF THE LUNG; ECHINOCOCCUS OF THE LUNG; ACTINOMYCOSIS OF THE LUNG.

By WILLIAM WHITWORTH GANNETT. M. D.

SYPHILIS OF THE LUNG.

From the anatomical standpoint there are two well-recognized and characteristic forms of syphilis of the lung about which there can be no question as to diagnosis—(1) the congenital form, and (2) the acquired form associated with the formation of gummata. Other changes in the lung are considered by many to be the result of acquired syphilis, but they are not absolutely characteristic.

(1) **Congenital syphilis** manifests itself in the lung in the form of the so-called white pneumonia, sometimes affecting a whole lung, sometimes present in large patches. The anatomical appearances are so striking that if once seen it can never be forgotten. The lung is large, does not retract, is very pale, almost grayish white, and of a firm consistency. The term white hepatization applied to it by Virchow gives an excellent idea of its appearance. Microscopically, the changes are those of a diffuse round cell growth in the alveolar walls, leading to thickening of the same and encroachment upon alveolar spaces, with proliferation and desquamation of the alveolar endothelium, thus filling the alveolar spaces. These cells contain numerous fat drops, the protoplasm having undergone fatty degeneration.

This form of lung may be found in children born dead or dying very shortly after birth. It may also be present in cases developing the signs of congenital syphilis a few weeks after birth.

The symptoms referable to the lung are very indefinite, though physical examination shows the signs of consolidation of a varying portion of the lung. There are likely to be manifestations of syphilis elsewhere in the body, so that the practitioner is not obliged to decide the case as being one of syphilis by the pulmonary signs alone. In fact, it would be quite impossible to differentiate clinically between a white pneumonia and the ordinary broncho-pneumonia common in children.

The cases usually die, though they occasionally yield to treatment when the symptoms develop some weeks after birth.

(2) **Acquired Syphilis.**—Pathological Anatomy.—The changes in the lung due to acquired syphilis manifest themselves in the form of gummata and more or less diffuse interstitial lesions. About the latter there is always a question of diagnosis even at the autopsy. Gummata, on the contrary, are quite characteristic. They are, however, of extreme

rarity. As elsewhere in the body, they are present in the form of distinct firm nodules, varying in size from a small pea to a peach, with a characteristic pale yellow, cheesy centre, of firm consistency, surrounded by a softer, more translucent gray tissue, to the outside of which is an area of injected lung tissue.

These gummata are likely to be situated near the root of the lung, either in the immediate neighborhood of, or else directly connected with, bronchi. In this way they may cause symptoms by pressure. Unless pressure symptoms are present leading to dyspnœa, gummata of the lung are not recognized clinically, the diagnosis being made at the autopsy.

Of the other anatomical changes which may possibly be of syphilitic origin there are to be mentioned the interstitial changes in the lung associated with the growth of dense connective tissue. Of these forms the one most suggestive of syphilis is that starting at the root of the lung and extending along the larger bronchi. The retraction of the connective tissue in its change from a young to a dense scar tissue may lead to stenosis of the bronchi. There may be growth of connective tissue in the bronchial walls and in the peribronchial tissue—that is, peribronchitis—forming often nodular masses. Secondary to this peribronchitis there may be broncho-pneumonia.

The connective tissue may be irregularly distributed in the lung in the form of bands, or even masses, replacing the lung substance. The bronchi of this part often undergo dilatation, forming a cavity. Lastly, syphilis may lead to interstitial pneumonia, as described under that heading.

The pulmonary lesions of syphilis occur in the late stages of this disease, and are usually associated with characteristic manifestations elsewhere in the body. In the absence of signs other than in the lungs the diagnosis of syphilitic lesions of the lung becomes exceedingly difficult if not impossible.

Symptoms.—When such confirmatory evidence apart from the lung is present, it is reasonable to suspect that symptoms and signs connected with the respiratory apparatus may be due to syphilitic changes. But this conclusion is to be reached by eliminating other conditions rather than by finding positive indications, especially excluding tuberculosis by bacterial examination of sputa, tumors, and aortic aneurysm.

Previous to the time when tuberculosis could be diagnosticated by finding tubercle bacilli in the sputa it is probable that cases of tubercular disease of the lung were mistaken sometimes for syphilis. At least the reports of cases of alleged syphilis of the lung give one this impression.

The most common and, at the same time, most persistent symptom of syphilis of the lung is dyspnœa, increasing as the disease advances and intensified by exercise. Cough is frequently present, sometimes without expectoration, sometimes with muco-purulent sputa.

The *physical signs* vary according to the character of the lesion, being those of bronchial stenosis or bronchiectasis, according as predominant narrowing or dilatation of bronchi exists; or the signs may be those resulting from bronchitis or from interstitial pneumonia. In nearly all cases the signs are obscure, and one must rely largely, in making a diagnosis, upon the lesions elsewhere in the body.

The PROGNOSIS is unfavorable, as a rule, death resulting from disturbance in breathing or from exhaustion.

TREATMENT with iodide of potassium and mercury should be tried in all cases where, other diseases being eliminated, syphilis is suspected. (See Treatment of Syphilis, Vol. I. p. 895.)

NEW GROWTHS OF THE LUNG.

VARIETIES.—New growths of the lung proper are of relatively slight importance, far less so than those of the pleura or mediastinum, with which, clinically, they may be confounded.

Tumors of the lung are rarely primary. Usually they represent a secondary development through infection, by transfer of bits of a primary growth elsewhere in the body, such bits being transferred by the blood currents through veins. The lung may also be the seat of malignant tumors due to a direct extension of the growth from surrounding parts, as pleura, mediastinum, or œsophagus.

The majority of the secondary growths are cancerous, usually in the form of multiple small nodules scattered through both lungs. In most cases their presence gives rise to no clinical symptoms or signs.

Next in frequency of secondary growths are the sarcomata. These occur in two forms—either metastatic nodules from some primary growth situated almost anywhere in the body, or else the lympho-sarcoma secondary to similar diseases in bronchial or mediastinal lymph-glands.

Enchondromata occasionally occur as secondary growths.

When the malignant disease originates in the pleura or mediastinum the symptoms and signs are referable almost wholly to the affection of these parts, the secondary development in the lung being of relatively slight importance.

When secondary nodules of cancer or sarcoma reach a considerable size, they may give rise to symptoms attracting attention to the chest. In such cases the existence of the primary growth elsewhere in the body has usually been manifest for some time, so that the mere fact of occurrence of pulmonary symptoms would suggest to the practitioner the probability of secondary development in the lung.

Of primary growths in the lung, cancer is the most common, sarcoma next, enchondroma least frequent.

Primary cancer, as a rule, is limited to one lung, though secondary nodules from this growth may be found in both lungs. It is always a cylindrical cell cancer, showing that it originates from the bronchial mucosa. It occurs in those past middle life, involving the upper lobe more commonly than the lower, and is more frequent in the right than in the left lung. It tends to spread through the lung tissue, forming a mass of a grayish yellow color and soft consistency. The tendency is for it to involve the pleura and neighboring lymph glands.

Primary enchondroma takes its origin from the bronchial cartilages.

SYMPTOMS.—Symptoms of new growths in the lung depend partly upon the size, but more especially upon the situation, of the nodules.

Pressure of the tumor upon a bronchus or extension of the growth to pleura, with the associated pleurisy and effusion, is common.

The usual complaint on the part of the patient is difficulty in breathing, with a sense of weight in the chest, increasing in time to severe dyspnœa. Cough is a frequent symptom, often paroxysmal in character. The expectoration may be ordinary mucus, or it may resemble prune-juice from admixture with blood. Occasionally hæmoptysis occurs, due to erosion of a vessel in the new growth.

In some cases patients complain of difficulty in swallowing, resulting from pressure of the tumor on the œsophagus, or the growth may be so situated as to compress the recurrent laryngeal nerve, leading to hoarseness of the voice.

Occasional neuralgia in the distribution of the brachial plexus is present as an indication of pressure of the tumor. Trachea or bronchus may be narrowed in the same way. Pain of a stitch-like character may indicate involvement of pleura with circumscribed pleurisy.

Physical examination may show in the case of large growths a bulge of the chest (independent of associated pleuritic effusion). On percussion, dulness, with sense of loss of elasticity to the pleximeter finger, is usually present. Auscultation shows either bronchial or absent respiration, often with râles. These signs are not, of course, diagnostic of a new growth, but if a tumor is suspected they are of value in localizing it.

Enlargement of glands in the axilla, dilatation of veins in the neck from intra-thoracic pressure, are of value in the confirmation of the diagnosis.

One of the commonest complications of new growths in the lung is extension to the pleura, with consecutive pleurisy and effusion. This fluid is nearly always hemorrhagic.

Associated with malignant disease of the lung are the general signs common to cancer developing in any part of the body—anæmia, cachexia, gradual failure of flesh and strength.

The DURATION varies from six to eighteen months.

As to TREATMENT, very few cases are amenable to surgical interference. Other means are, at present, lacking.

ECHINOCOCCUS OF THE LUNG.

ECHINOCOCCUS—or hydatid cyst, as it is frequently called—occurs occasionally in the lung, but it very rarely originates in this organ. Far more commonly it represents the result of an extension of an echinococcus of the liver which has ruptured through the diaphragm.

As is well known, the echinococcus cyst represents a mid-stage in the course of development of the worm known as the Tænia echinococcus, which lives in the intestines of the dog. The eggs discharged in the fæces of the dog, when taken into the human being, as happens frequently in Iceland, where dogs and men occupy the same dwelling, develop this cyst, which may reach the size of a man's head, and is

filled to a greater or less extent with smaller daughter cysts developing from the inner wall, and fluid. These cysts may become obsolete, or suppurative processes may occur in them, and in the latter case they usually break through the tissues surrounding them and discharge their contents. If the cyst is situated in the lung, the rupture may occur into a bronchus or into the pleural cavity.

Symptoms due to the presence of an echinococcus cyst in the lung may or may not be present. Even when present they are not especially indicative of a cyst, being pain, cough, and sometimes expectoration.

Physical examination would give dulness and diminished respiratory murmur.

Diagnosis.—The diagnosis is usually made when the patient expectorates the contents of the cyst, microscopic examination of which shows the characteristic hooklets or the membrane forming the wall.

When the discharge occurs into the pleural cavity, acute pleurisy, usually purulent, follows, leading to opening artificially. The peculiar bits of membrane floating in the fluid attract attention and suggest the desirability of a microscopic examination, which reveals the true nature of the disease.

Recovery sometimes takes place when the contents of the cyst are coughed up, or when the cyst is so situated as to make it amenable to surgical interference.

One of the greatest dangers is, that the cyst become gangrenous or suppurate, in which case by extension to neighboring parts, as peritoneum or pericardium, a fatal result is likely to follow.

Treatment.—Attempts to destroy the parasite by inhalations, of substances like turpentine, have proved of no avail, nor is any medical treatment at present known efficient. Surgical treatment, on the contrary, is occasionally successful.

ACTINOMYCOSIS OF THE LUNG.

Definition.—Under the general heading "actinomycosis" is understood a series of changes associated with new growths belonging to the granulomata, which have a tendency to soften and suppurate, and in which is found a characteristic organism occurring in colonies, the central part made up of a tangle of threads, the peripheral part having club-shaped bodies arranged in the radii of a circle, hence called by the botanist Hartz ray-fungus or actinomyces.

Pathology.—Actinomycosis was first observed, and its relation to the ray fungus accurately described, by Bollinger in 1877, who noticed the growth in the form of a tumor in the jaw of cattle. Section of the growth showed the presence of sulphur yellow bodies in the mass, which microscopically proved to be clumps of the peculiar fungus described above. In 1878, J. Israel of Berlin described a case occurring in man, and pointed out the causative relation of the fungus to the growth; and Ponfick in his monograph, published in 1882, established the identity of the disease in man and animals.

In man, as in animals, growths may occur in the jaw, probably by infection through a carious tooth, from which secondary growths in the neck may result, or infection may occur through the pharynx. This form is of interest to the surgeon. But development in the internal organs, notably lung and pleura, may take place, thus rendering the disease of interest to the physician.

When lung or pleura is involved, the infection may be secondary to the neck or jaw, or it may result from a primary infection through the bronchi, usually the latter.

In certain cases prevertebral abscesses form with gravitation of the pus, and so infection of mediastinum, lung, or pleura, but this is much less common than is infection through bronchi.

When the infection is by the bronchi, there may result a bronchitis, with cough and expectoration of a muco-purulent fluid of disagreeable odor, and in which actinomyces are found. In fact, it is only by finding these that the diagnosis is made.

The bronchitis may go on to the development of broncho-pneumonia, but this is of the peribronchial type; that is, the inflammation begins in the bronchial walls and extends to the peribronchial tissues, with pneumonia developing about them. Not only is there an inflammatory product, but there is a new growth of connective tissue, tending to render the part of the lung affected still more solid. Extension to the pleura, with adhesive inflammation between the two layers, with circumscribed collections of pus in spaces between layers of pleura not united, may result. The pus may perforate the chest wall or there may be inflammatory process in the chest wall, with marked induration and thickening of the skin, with later perforation externally, and erosion of sternum or ribs may occur.

This tendency of actinomycosis starting in the lung to make its way outward and involve pleura, chest wall, and skin is frequently noted in man.

The actinomycosis proper is associated with the production of a granulation tissue, which tends to undergo necrosis, thus forming a rather soft, whitish, pultaceous mass. If hemorrhage has occurred during the softening process, the material becomes brown. The suppuration so common in actinomycosis is probably dependent upon pus-producing micrococci, and not upon the actinomyces.

Clinically, pulmonary actinomycosis presents itself either in the form of bronchitis, or broncho-pneumonia, or empyema, or as a more or less extensive peripleuritic infiltration, giving rise to marked thickening and induration of the chest wall, with burrowing of pus through the subcutaneous tissues, forming pockets of pus connected with each other by irregular fistulous tracts. Later, the pus may make its way through the skin, discharging externally. The process is a slow, insidious one, but progressive.

Symptoms.—The symptoms may be those of a bronchitis or broncho-pneumonia. In the former case the expectoration, as previously mentioned, is usually somewhat foul, and shows microscopically the characteristic ray-fungus. In the broncho-pneumonic variety the signs are those of consolidation of the lung, but the symptoms are those of progressive emaciation and hectic fever of pyæmic character when the

suppuration is developed. Later, the symptoms and signs are those of empyema, usually encapsulated, which leads the physician to tap the chest. Finding pus, it should be in all cases examined for actinomyces. When the process has become peripleuritic, with infiltration of chest wall, abscesses, and often fistulæ externally, it presents an appearance still more suggestive of actinomycosis, and, although frequently mistaken for tuberculosis, yet the means of differential diagnosis are readily at hand in the examination of the pus.

The actinomyces can be made out readily in pus oozing from a fistulous opening. Although different parts of an actual colony of actinomyces show variations in structure, the central parts being more filamentous and tangled, with branching filaments about them and the clubs in the peripheral part, yet for diagnosis in pus one relies especially on the club- or pear-shaped forms. The variation in shape depends upon the age of the actinomyces, the clubs being seen in the later stage of development of a colony, very likely due, as mantained by Bostroem, to a degeneration in the sheath of the organism. General opinion now is that this organism belongs among the polymorphous bacteria, since in a growing colony there may be found round bodies resembling cocci, also longer and shorter rods like bacilli, also threads which may be single or branching. The club-shaped bodies, so important in aiding in the diagnosis, are transparent, and in them may be found cocci and rods.

In the tissues actinomyces are best stained by the Gram method.

The PROGNOSIS in actinomycosis is unfavorable, for at present no satisfactory means of treatment is known. When empyema or infiltration of the chest wall with suppuration has occurred, surgical means may be tried.

Death results from the infection, with exhaustion.

THE NON-TUBERCULAR DISEASES OF THE PLEURA.

By HERBERT B. WHITNEY, M. D.

PLEURISY.

Definition.—Pleurisy is an acute or chronic inflammation of the whole or a part of the pleural membrane, characterized by the formation in the pleural cavity of a fibrinous, sero-fibrinous, or purulent exudation.

The classification of the many forms of pleurisy on a strictly scientific basis presents serious difficulties, and the one which we adopt is based solely on certain sharp clinical distinctions which seem to be of practical value in the recognition, prognosis, and treatment of this disease.

Fibrinous or Dry Pleurisy.

Etiology.—Certain cases of dry pleurisy are difficult to explain except on the supposition of a primary form. Some of these appear to be the result of sudden or prolonged exposure to cold. Others are to be regarded as a manifestation of the rheumatic diathesis—possibly also, in rare instances, of syphilis. Traumatic forms may also be reckoned here, such as follow blows or compression.

Most cases of dry pleurisy are secondary, usually, to some affection of the lung. Without enumerating all the inflammatory conditions of neighboring organs which might possibly be complicated with dry pleurisy, it will be sufficient here to indicate those in which this form is a common occurrence. Chief among these is pulmonary tuberculosis. Not only are evidences of pleurisy rarely missed in persons who have died of this disease, but it would also appear that a majority of all cases of pleural adhesions develop in phthisical subjects. Smith[1] found post-mortem evidences of tuberculosis in 68 per cent. of 140 cases of pleural adhesions: undoubtedly most of these were the result of dry pleurisy, since the latter is far more frequent in phthisis than pleurisy with effusion. It does not, however, follow that a pleurisy which is secondary to phthisis is itself tubercular; Smith found tubercles in only 9 of his 140 cases. Other diseases of the lung usually attended by dry pleurisy are the various forms of pneumonia and infarctions. It is also not an infrequent complication of pericarditis. It may accompany the various infectious diseases, and, in general, it would appear that any one of the numerous causes of sero-fibrinous pleurisy may at times produce only the dry form. Whether—as, for example, in typhoid—such a pleurisy

[1] *Med. News*, 1890.

is a primary microbic affection or merely secondary to some such affection of the lung is a question to which no answer has yet been given.

Pathological Anatomy.—A recent case of dry pleurisy is usually of very limited extent, although the disease may successively invade large portions of the pleura. Experiments upon animals have shown the following pathological changes: There is first a localized congestion of the pleura, with some loosening of its epithelial layer, and moderate increase of leucocytes in the subserous connective tissue. Its surface, therefore, is reddened, and in a few hours it loses its glistening appearance as a result of the beginning free exudation of lymph. The fixed connective tissue cells soon show signs of increased activity; they are found in active cell division, and thrust themselves in groups through the epithelial layer into the surface coating of lymph. The latter meanwhile has increased in thickness; by the fourth or fifth day it already contains newly formed capillaries which have developed from the connective tissue cells, and later still it becomes organized into fibrous tissue. Usually, therefore, there is afterward no complete *restitutio ad integrum*, but there remains at least a small bluish white area of pleural thickening. In other cases the exudation of lymph is somewhat more abundant; the opposite pleural surfaces agglutinate, and eventually become firmly united by the permanent organization of the connecting lymph into cicatricial tissue.

Symptoms and Course.—Dry pleurisy may be either acute or of slow and insidious development. The acute form begins with a sharp cutting pain in the side, usually referred to the affected region, but sometimes felt below in the abdomen or even on the opposite side of the chest. An initial chill is very rare, but there is usually some fever, which on the first day may amount to 102° or 103° F.: on the following day, however, it is less, and it very commonly drops to normal on the third. Respiratory symptoms are mild. The breathing may be considerably accelerated because less painful when superficial, and there is often a dry, catchy cough. The pain itself is usually of short duration, and after twenty-four to thirty-six hours it subsides to a feeling of soreness, which in turn gradually disappears. This is the course which most primary dry pleurisies are likely to pursue, and the usual termination is in complete recovery.

In a few cases, however, the disease has a tendency either to repeatedly recur or to advance by a slow and almost painless course to a condition of great pleural thickening, with secondary fibroid invasion of the lung. Most of these cases are probably tubercular, although the argument of Sir Andrew Clark[1] to the contrary is difficult to resist, and we regard the question of a non-tubercular form of fibroid phthisis originating in a dry pleurisy as still undetermined. In other cases the only result of the recurrent pleurisy is the formation of universal adhesions between the visceral and parietal pleuræ. This synechia, however, is not always so harmless as it might appear: the consequent impairment of pulmonary capacity seems to favor the development of a chronic bronchial catarrh, which may in time lead to circulatory stasis and death.

The symptoms of secondary dry pleurisy are apt to be very mild. In pneumonia they are overshadowed by the primary disease. Occur-

[1] *Lancet*, 1885.

ring in the course of pulmonary phthisis, dry pleurisy sometimes causes an ephemeral rise of temperature, but is usually indicated only by fleeting pains about the scapula or shoulder. A mild character is not, however, peculiar to secondary forms, and it is probable that many individuals in apparent health have occasional light forms of pleurisy, which cause at most a mere feeling of pressure or uneasiness.

PHYSICAL SIGNS.—The physical signs of acute dry pleurisy are few. The respiratory movement may be somewhat diminished, but practically the only important sign of this disease is friction. Its most typical form is a soft grazing rub, which may be closely imitated by pressing one hand tightly over the ear and drawing a finger of the other back and forth across its surface: occasionally the sound is leathery and creaking. It is heard oftenest with both inspiration and expiration, is unaffected by cough, and may sometimes be perceptible on palpation. It is a notable fact that but little relation appears to exist between the extent or intensity of friction and the severity of pain; it is common enough to discover a wide area of intense friction in cases where there is absolutely no pain or even soreness. In the form of dry pleurisy attended with chronic fibroid thickening there is often marked retraction of the chest wall, with perhaps some drooping of the shoulders, projection of the scapula, and lateral curvature of the spine. On percussion resonance is found to be nearly or quite absent over the lower portions of the chest, and there is greatly diminished voice, respiration, and fremitus. Some authors describe also a sort of pleural râle in these cases—a crackling, creaking, or "rustling" sound of such superficial character that its pleural origin is apparent. The diagnosis of dry pleurisy from intercostal neuralgia or pleurodynia is sometimes aided by the presence of fever; usually it must be based almost solely on the discovery of friction.

A friction sound is sometimes heard in pleurisy which is synchronous with the action of the heart. It is due to the rubbing of the roughened pericardial against the pulmonary pleura, and is to be distinguished from ordinary pericardial friction by its intensification at the height of inspiration, and by its limitation to the left border of the præcordial area.

SERO-FIBRINOUS PLEURISY.

ETIOLOGY.—The etiology of pleuritic effusion may be said to be undergoing at the present time a marked process of evolution. It is not only the object of extensive bacteriological research, but clinical investigation appears to have received a fresh impulse, and many facts have recently come to light which are of the most far-reaching significance. Among the most striking of these is the relation of sero-fibrinous pleurisy to tuberculosis. This matter is so important that it may be well to consider briefly some of the various sources of evidence.

1. *Personal and Family History.*—The evidence here is largely negative. Moreover, available statistics are so meagre and so difficult to interpret correctly that their value is very small. Lindsay[1] found a family or personal history of tuberculosis in only 5 of 38 cases of pleurisy; among 58 cases investigated by Osler[2] there was a family

[1] *Lancet*, 1892.

[2] *Boston Med. and Surg. Journ.*, 1893.

history of tuberculosis in 19, and in the same number reported by Sittman[1] only 14. These figures prove but little on either side.

2. *The Future of Pleuritics.*—It is upon the fact that a considerable proportion of those who have had sero-fibrinous pleurisy eventually develop some form of tuberculosis that the belief in the frequent relationship between these affections mainly rests. It must be insisted upon that negative statistics are not necessarily of any value; the period of observation may have been too short. A single series of cases carefully followed for a long period of years is more convincing than any amount of testimony based on brief records or general impressions. A few trustworthy reports may here be adduced: V. Y. Bowditch[2] ascertained in 1889 the present condition of 90 cases of sero-fibrinous pleurisy which had been under the care of the elder Bowditch from 1849 to 1879; of these, 32 had become tuberculous. Ricochon[3] followed for thirteen years 32 private cases of sero-fibrinous pleurisy; he tells us that tuberculosis developed in all but 2. Barrs[4] found that of 62 cases treated at the infirmary of Leeds from 1880 to 1884, 32 had died in 1890, and of these 18 of phthisis and 4 of some other tubercular disease. Sears[5] has recently averaged the percentage of subsequent tuberculosis in eight series of cases reported by different observers; there was a total of 451 cases of pleurisy, with 176 deaths from tuberculosis—an average of 39 per cent.

These statistics seem to need no comment. It should, however, be borne in mind that the frequency of tuberculosis following pleurisy is no proof that the pleurisy itself is tubercular.

3. *Bacteriology.*—Microscopical and culture examinations of sero-fibrinous exudations for the tubercle bacillus are almost invariably negative: this is universally admitted. Inoculation with the exudate has been more successful in the few instances where it has been tried. Pansini inoculated from 15 cases and obtained positive results in 6. Those of Netter[6] were similar; positive results were obtained in 10 out of 25 cases of primary pleurisy. Netter also inoculated from 12 cases of evidently tubercular pleurisy, and only 7 of these were successful. It would therefore appear, from these observations, that 40 per cent. of all cases of primary pleurisy will produce tuberculosis by inoculation, while even if all were tubercular, only about 60 per cent. of inoculations could be expected to prove successful.

4. *Post-mortem Examination.*—Here we encounter great difference of opinion. Kelsch and Vaillard[7] claim to have found tubercular manifestations in the pleuræ of all cases of primary sero-fibrinous pleurisy examined. Osler,[8] on the contrary, found pleural tuberculosis in only 32 out of 101 cases of the various forms of effusion.

It seems, therefore, from these different investigations, that at least 40 per cent. of all individuals with sero-fibrinous effusion will eventually develop tuberculosis. More than this: the results of inoculation and post-mortem examination appear to confirm the belief that a large proportion of such pleurisies are of genuinely tubercular character. Just

[1] Quoted by Netter.
[2] *Trans. Am. Climatolog. Soc.*, 1890.
[3] *Rev. des Sciences méd.*, 1888.
[4] *Brit. Med. Journ.*, 1890.
[5] *Boston Med. and Surg. Journ.*, 1892.
[6] *Traité de Médecine*, Paris, 1893.
[7] *Arch. de Physiologie*, 1886.
[8] *Boston Med. and Surg. Journ.*, 1893.

how the bacilli gain access to the pleura is not quite clear, although in many cases there were probably pre-existing tubercular nodules in the lung or bronchial glands. These facts greatly modify our former ideas as to the origin of acute pleurisy; and, while cold draughts during perspiration or prolonged exposure to a chilling atmosphere must still be reckoned among the possible causes of this disease, it is probable that they are much oftener exciting causes than truly primary. We may add that good authorities are not wanting who wholly deny the etiological influence of cold in pleurisy.

Pleurisies which are, strictly speaking, of primary character may also be related to other conditions than tuberculosis. The pneumococcus has been occasionally found in sero-fibrinous effusions; most of these cases were complications of pneumonia, but a few have been entirely independent of any pulmonary affection. Acute inflammatory rheumatism may be immediately preceded or accompanied by acute pleurisy, or, in other cases, the latter may apparently be the sole manifestation of the disease. French and German writers consider rheumatic pleurisy very frequent: in my experience any evident association of the two affections is quite unusual. Sero-fibrinous pleurisy may furthermore be a manifestation of syphilis. Chantemesse and Widal first called attention to a variety which may accompany the roseola; Hutchinson has frequently encountered the affection in hereditary syphilis, and considers it a common cause of death; Pretorius and Talamon[1] have recently asserted that tertiary forms of pleurisy are not so very infrequent, and are rapidly influenced by the administration of the iodides. The Eberth bacillus of typhoid has been demonstrated in sero-fibrinous effusions by Sahli, Fernet, Bozzolo, and others, occasionally in pure culture; these effusions are usually associated with typhoid fever, but in a case recently reported by Charrin and Roger[2] there were the general symptoms of typhoid without, as the post-mortem showed, any intestinal lesions. Sero-fibrinous pleurisy occasionally accompanies the infectious diseases of childhood, though here the purulent form is much more frequent; possibly in either case the pleurisy is secondary to the bronchopneumonia which often complicates these affections. Traumatic forms may be mentioned here, such as may follow any external wound whether superficial or penetrating, or contusions with fracture of a rib or other deep-seated injury.

Purely secondary forms of sero-fibrinous pleurisy, aside from the meta-pneumonic, are oftenest terminal complications of chronic affections of the heart or kidneys. With the former the pleurisy is generally caused by an embolic infarction of the lung; in Bright's disease it has been attributed both to retained irritants in the blood and to increased susceptibility to cold and tuberculosis. Other occasional causes of effusion are parietal affections, aneurysm, new growths and necrotic processes in the lung, and affections of the abdominal cavity, such as peritonitis, and especially abscess and hydatids of the liver. Demons[3] has also called attention to the relative frequency of sero-fibrinous pleurisy with ovarian cysts; he has met with 9 instances in 50 cases.

Sero-fibrinous pleurisy is most common between the twentieth and

[1] *La Méd. moderne*, 1891. [2] *Soc. méd. des Hôp.*, 1891.
[3] *Bull. de la Soc. de Chir.*, 1887.

fortieth years of life, although it is a disease of all ages, and its occurrence, especially in childhood, and even in early infancy, is not exceptional. It is somewhat more frequent in men than in women: hospital statistics, which indicate a very great preponderance in men, are misleading. The disease appears to be somewhat more prevalent in the cold season—according to Engster, in January and April.

Pathological Anatomy.—The early changes of sero-fibrinous pleurisy do not differ perceptibly from those of the dry form, except that a much larger area is usually involved. With the development of effusion we have to do with the position and character of the fluid, the various phenomena of displacement, and the resulting changes in the pleura itself.

The position assumed by the fluid is dependent upon (1) its weight. The relative importance of this factor is difficult to determine. It is held by most German authorities to be predominant, by many American to be inferior to other forces. The experiments of Garland[1] would seem to prove that the fluid must occupy the lower portion of the pleural cavity in whatever position of the living animal it is injected. Dwight,[2] on the contrary, in frozen sections of the thorax of a child with effusion, who had died with head and shoulders below the horizontal, found that the fluid had gravitated to the upper region of the chest. Clinically, the base of the chest being always lowest, the result of physical examination is inconclusive as to the point in question. All admit that the weight of the effusion is an important factor in the displacement of organs. (2) The retractile force of the lung. It is of course well known that what is commonly called the cavity of the pleura is not an actual vacuum; the atmospheric pressure upon the outer thoracic wall, on the lower surface of the diaphragm, and within the lung forces these various parts into close apposition, the lung yielding most because it is most elastic. The normal position of the lung is therefore maintained in spite of its elasticity—a force which if it were not more than offset by the intra-pulmonary atmospheric pressure, would contract the lung to not more than one third its usual volume. This force is spoken of as the retractile force of the lung, and a like force is also inherent, to a certain degree, in the diaphragm and thoracic wall, which are likewise held by external atmospheric pressure in a condition of constant tension. It is therefore evident that the pleural cavity is normally under negative pressure, or, in common parlance, that the lung, diaphragm, and thoracic wall exercise a constant power of suction, which, the moment this cavity communicates either directly or through a column of water with the outer air, will operate to forcibly draw into it a certain quantity of air or fluid as the case may be. That this is what occurs under such conditions is seen in any case of pneumo-thorax, and Garland has demonstrated a like action upon fluids by experiment upon living animals. But, further, since the lung is manifestly not compressed by the fluid or air thus aspirated, since, on the contrary, these simply follow its retraction as water follows the piston of a pump, it is also evident that the shape which the lung assumes will depend mainly, not upon the presence of the fluid, but upon inherent conditions of pulmonary elasticity. Those parts will naturally retract most and with the greatest

[1] *Pneumodynamics*, Boston, 1878. [2] *Bost. Med. and Surg. Journ.*, 1882.

force which are in a state of the greatest tension—whose radii of distention are longest. It could therefore, on a priori grounds, hardly be expected that the upper level of fluid would be horizontal, since this would imply a high degree of pulmonary retraction along certain radii and little or none along others; and, in fact, both experimental and clinical evidence demonstrate conclusively that the fluid of simple effusion does not assume a hydrostatic level. On the contrary, it simply occupies the irregular space left vacant by the retracting lung and, to a lesser degree, by the retracting diaphragm and thoracic wall. The fluid, in short, accommodates itself to the lung, and not the lung to the fluid, except in so far as the weight of the latter is not an absolutely indifferent factor. We find, therefore, in effusion, corresponding to these deductions, that the column of fluid is generally shallowest at the median line and deepest in the region of the axilla. For establishing these facts upon a scientific basis we are chiefly indebted to G. W. Garland.

It seems probable that still another factor has a certain influence upon the shape and position of the fluid— *i. e.* (3) a certain degree of cohesion which exists between the pulmonary and costal pleuræ. This has been demonstrated by the observations of West [1] in connection with traumatic and artificial pneumo-thorax.

It must be admitted that in any individual case the relative importance of these three factors would be very difficult to estimate. It is evident that as an effusion increases its weight becomes a factor of constantly increasing importance, while that of pulmonary retraction grows correspondingly less. Moreover, both may vary in different cases. A purulent exudation is heavier than a serous; and not only is the elasticity of some lungs normally greater than that of others, but it may be variously impaired by disease. As to the exact shape assumed by the fluid, this is a matter of especial clinical interest, and will therefore be discussed in connection with the physical signs. An effusion may be limited by adhesions in all conceivable ways: it may occupy several compartments which may or may not communicate, or it may exceptionally fill the meshes of several superimposed fibrinous layers.

The character of the exudation does not differ in the main from that of diluted blood serum. It is a pale yellow, or in older cases more brownish yellow, odorless fluid, usually transparent, sometimes slightly opaque: macroscopically, there is no sharp line of distinction between a sero-fibrinous and a purulent effusion. In the fluid is usually suspended a varying quantity of fibrinous flocculi, and it often coagulates spontaneously four to twenty-four hours after withdrawal. On chemical examination it is found to be alkaline. It always contains in solution a considerable quantity of fibrin and albumin, the latter varying with the acuteness of the process, but usually amounting to 1.0 or 1.50 per cent. Its mineral constituents are similar to those of serum. It may in certain cases contain glycogen, urea, uric acid, and the coloring matters of the bile; also cholesterin and free fat, the latter especially in chronic cases. Microscopically, the presence of leucocytes is found to be constant, with here and there an epithelial cell. Red corpuscles are only occasionally encountered, oftenest in tuberculosis and new growths of

[1] *Brit. Med. Journ.*, 1887.

the pleura and in cases of purpura or other forms of the hemorrhagic diathesis. According to Dieulafoy, at least 6000 red disks to the cubic centimetre are necessary in order to impart the faintest rose tint to the fluid; larger quantities may produce a pronounced red color of varying intensity. Other occasional microscopical constituents are cholesterine crystals, fat globules, and the various bacteria referred to in the section upon Etiology (pp. 260, 261).

The displacement of organs which results from an effusion must of course vary greatly with the amount of fluid. We have already seen that a considerable quantity is necessary (1800 to 2400 c. c., Weil)[1] before its tension becomes positive and actual compression of neighboring parts begins. It would, however, be an error to suppose that displacement of organs other than the lung does not begin until an effusion has reached a considerable size. We have seen that not only the lung, but also the diaphragm and thoracic wall, are normally maintained by the external atmospheric pressure in a state of constant tension. If, now, the negative pressure of the pleural cavity is diminished, as it necessarily must be, by the entrance of even a minimum quantity of fluid, a corresponding degree of relaxation must ensue, not only of the lung, but also of the diaphragm and thoracic wall; while the mediastinum, having now a less negative pressure on one side than on the other, will inevitably move in the direction of least resistence—namely, toward the well side. And this corresponds also both with the result of Weil's[2] experiments in pneumo-thorax and with the clinical fact that in left-sided effusion the heart is found displaced long before actual compression can have possibly begun. We find, then, in sero-fibrinous pleurisy all grades of displacement of the various organs involved. The lung itself may be only partially retracted, with possibly a limited atelectasis of its lower borders, or it may be compressed against the spine to an airless fleshy mass: in cases of long standing it may present interstitial changes as a result of direct extension of inflammation from the pleura. The mediastinum may be at or beyond either margin of the sternum. The heart is usually displaced en masse, the relative position of base and apex remaining unchanged. In right effusion its apex may be an inch or more outside the left mammary line and slightly elevated. With effusion into the left pleura cardiac displacement is much more pronounced, the apex of the heart being then often found in the median line and its base near the right nipple: here there is also slight rotation of the heart upon its axis, and the apex is apparently slightly lowered. Very rarely, perhaps, the heart may be completely rotated, so that its apex lies beyond the right mammary line, although many experienced pathologists have never encountered this condition. Bartels and Fräntzel[3] claim to have also found in excessive displacement toward the right an angle or bend in the inferior vena cava where it leaves the diaphragm, sufficient to produce a veritable obstruction; by others the possibility of this is denied. The diaphragm may be depressed to such a degree that its convexity faces downward; its displacement is attended by a corresponding depression of the liver and spleen, and the former may also, in left-sided effusion, be tilted upon its antero-posterior axis,

[1] *Topographische Percussion.* [2] *Zur Lehre v. Pneumothorax*, reprint (1879).
[3] *V. Ziemssen's Handbuch*, "Pleuritis."

so that its right lobe is lower than the left. An effusion is also attended by slight depression of the diaphragm of the well side: evidently, with the aforesaid movement of the mediastinum there must be a corresponding relief in tension, not only of the diaphragm, but also of the lung and thoracic wall of the unaffected side.

The following changes may be observed in the pleura itself: Coincident with the effusion of serum there is a more or less abundant exudation of lymph which forms a pseudo-membranous coating upon the pleural surface. The effusion also partially coagulates into flocculi and clumps which are largely deposited upon the walls as a rough, irregular layer. Thus the whole pleura becomes invested with a false membrane of a yellowish white color, soft and friable in consistency, and varying in thickness from one sixteenth to one half of an inch. Meanwhile, the pleura beneath has become infiltrated with leucocytes, new capillaries have formed, and thus a bed of granulation tissue has developed which tends to push upward through the superimposed fibrinous layers. In favorable cases most of these inflammatory products are eventually absorbed, the fluid portion being rapidly taken up by the lymphatics, chiefly of the costal pleura, and the solid constituents and false membranes after a fatty degeneration which is of slower progress. The opposite pleural surfaces, however, usually remain united by permanent adhesions. In other cases, oftenest tubercular, absorption does not take place. There is frequent recrudescence of the inflammatory process, and the organization of successive deposits of lymph results in the formation of a dense cicatricial tissue which, a half inch or even more in thickness, may invest the whole costal and visceral pleura. The contraction of this fibroid tissue, and the inability of the lung to expand when after months or years the fluid is finally absorbed, often produce great deformity of the chest. Instead of its normal convexity, the side may present a well marked depression, with perhaps a consequent drooping of the shoulder, outward tilting of the scapula, and more or less lateral curvature of the spine: these retractions are much more likely to be local and circumscribed than to involve the whole half of the chest. Heart and mediastinum, unless adherent, are drawn toward the affected side, and the diaphragm assists also in filling the vacuum by a permanent upward displacement.

Calcareous masses have been found in such pleuræ sometimes several inches in length.

For a description of the tubercular processes usually associated with these chronic forms I refer to the article by Osler on Tuberculosis (Vol. I. page 767).

Symptoms.—Two forms of sero-fibrinous pleurisy may be regarded as more or less typical—the acute febrile and the latent.

The acute form is of sudden onset, with only exceptionally slight prodromata of one or two days' duration. Chilly sensations may usher in the attack, but a well-defined rigor is rare. In the great majority of cases the first and for several days most prominent symptom is a severe pain in the side. This pain is usually sharp and lancinating in character, more rarely boring, tearing, or pressing; sometimes it is dull at the outset, and does not attain its maximum of intensity for twenty-four to forty-eight hours. Its most frequent situation is in the fifth or sixth

space just outside the nipple, but it may be felt in any part of or even throughout the chest, and exceptionally in the abdomen, or even on the well side. It is aggravated by cough, deep inspiration, and pressure. Its period of greatest intensity corresponds in general to the dry stage, and is of from two to five days' duration; with the development of effusion it usually subsides into a more diffused ache or suddenly disappears. Pain is much less pronounced in the aged and in those debilitated by disease; in very rare instances it may be entirely wanting. With the onset of pain there is a rise of temperature, usually to 102° or 103° F., and attended by a corresponding frequency of the pulse. As a rule, this fever continues, with morning remissions, for ten to fourteen days, when it gradually falls to normal: occasionally it persists with diminished intensity until the effusion itself disappears. There is nearly always a varying degree of dyspnœa from the first. It is usually not intense before effusion, and is limited to a moderate acceleration of breathing due both to the fever and to the shallowness of respiration necessitated by the pain. It is said to be abdominal in type in costo-pulmonary inflammations, costal in diaphragmatic. Cough is rarely wanting at some period of the disease: it is short, painful, suppressed, and usually unaccompanied by expectoration. The position assumed by the patient is somewhat characteristic, and is governed by the painfulness of respiratory movement and of pressure upon the inflamed region. At first he either lies upon the well side to avoid pressure, or takes a semi-recumbent posture with slight rotation toward the affected side, thus combining freedom from pressure with a certain degree of immobility. As the effusion accumulates and dyspnœa increases this position is reversed, in order that the respiratory movement of the well side may be unimpeded.

The effusion probably begins to form within a few hours from the onset of the disease: in the experiments of Andral and of Wintrich upon animals it followed the injection of irritants in five to thirty hours. Its effects, however, are plainly perceptible only when a considerable quantity has collected, oftenest perhaps from the second to the fifth day. The symptoms produced by effusion are chiefly those relating to the pulse and respiration. The latter is, in general, accelerated in proportion to the quantity of fluid and the resulting diminution of aërating surface in the lung. To this rule, however, there are many exceptions. A small effusion may sometimes cause excessive dyspnœa, while with a large one there may be none—differences to be accounted for by variations in the rapidity of exudation, in the condition of the lungs, and in the susceptibility of the respiratory centres, which is apt to be greatest in robust individuals. In some cases also extreme dyspnœa may be the result of complications which will be hereafter considered. The pulse is usually somewhat increased in frequency, even in small effusions and when there is no fever; with a large effusion it is usually not only rapid, but also small and weak. This latter effect is the result of a diminished arterial tension from pressure of the effusion upon the heart and great vessels—an interference with the circulation which in certain cases may cause sudden death. The effusion, as such, does not produce fever, although, as previously stated, the latter often continues until the effusion has disappeared. The general condition is disturbed in proportion

to the fever and dyspnœa; and independently of these there is also a marked tendency to progressive weakness and anæmia if the effusion persists beyond a certain period. Occasionally with large effusion there is troublesome vomiting, attributed by Ferber and Fräntzel to a dislocation of the stomach. The urine is usually scanty during the acute stage, of high specific gravity, and often containing a moderate quantity of albumin; with beginning subsidence of the effusion it rapidly increases, and soon reaches a daily quantity of 2000 to 3000 c. c. In excessive effusion with great displacement the urine may present all the characteristics of passive hyperæmia of the kidney.

The COURSE of acute primary sero-fibrinous pleurisy, though always uncertain, is not lacking in a certain tendency to a typical evolution. In at least a large number of cases the effusion increases steadily for a time, then for a few days is stationary—the "période d'état"—and finally subsides, at first rapidly, and then more slowly and steadily, to complete resorption. The exudative stage and that of absorption have each an average duration of perhaps fifteen days; the période d'état lasts usually from two to seven days—may, however, sometimes be wholly lacking. A case, therefore, of acute pleurisy with small or moderate effusion may be expected to last four to six weeks, to which at least another month must be added for the complete absorption of false membranes, and an approximate *restitutio ad integrum*. Purely rheumatic cases are said to be much shorter; according to Netter, the effusion forms in a few hours, and in from two to five days may be wholly reabsorbed. Very large effusions, on the other hand, if left to themselves, seldom disappear in the above-stated period, but show a decided tendency to chronicity.

In the second type of sero-fibrinous effusion—the so-called latent form—the affection is of insidious onset and more chronic course. Its beginning is often entirely unnoticed by the patient, and he first presents himself to the physician when an effusion of considerable size has developed. His chief complaint is of cough, or of shortness of breath especially on exertion, or, possibly, of some pain or uneasiness in the side. There is more or less depression of the general health; appetite is impaired, strength is diminished, and there is usually a considerable degree of pallor. In some cases, particularly in children, these general symptoms are the only ones which have been noticed, and there is absolutely nothing to call attention to the chest. These latent forms are especially common at the extremes of life, in individuals already weakened by other disease, in chronic affections of the heart and kidneys, and in tuberculosis.

Between the acute and latent forms of sero-fibrinous pleurisy there are all possible varieties and degrees. Many secondary pleurisies are of course overshadowed by the primary affection, and their symptoms pass unnoticed. Again, a pleurisy of vigorous onset may be apparently fully recovered from, and yet a latent form of inflammatory process be left behind which continues its slow evolution. In all forms months, or even years, may elapse before the effusion is spontaneously absorbed, or, if it is artificially withdrawn, it may persistently reappear. Probably a large proportion of these latent and chronic recurring forms are tubercular. They may, however, be fully recovered from, or, more com-

monly, unless the patient dies in the meanwhile of tuberculosis, there remains great fibroid thickening of the pleura, with subsequent retraction and often sclerotic processes in the lung itself—conditions which tend to consecutive disease of the circulatory apparatus and a steady impairment of the general health. Life may, however, be prolonged for a great many years.

The PHYSICAL SIGNS of sero-fibrinous pleurisy do not at first differ from those of the dry form, except, perhaps, in a more widespread occurrence of friction, corresponding to the greater severity of the inflammatory process.

The quantity of effusion necessary for the production of well marked physical signs in adults is not accurately known, but is usually estimated at from 300 to 500 c. c. It is possible that even these figures are too low. I recently chanced to make a very careful examination of an individual who five minutes afterward suddenly dropped dead: although the examination showed unimpaired resonance on both sides behind to the level of the eleventh dorsal vertebra, the autopsy discovered 750 c. c. of fluid in the right pleural cavity. That this was not a post-mortem transudation is measurably assured by the fact that it was unilateral.

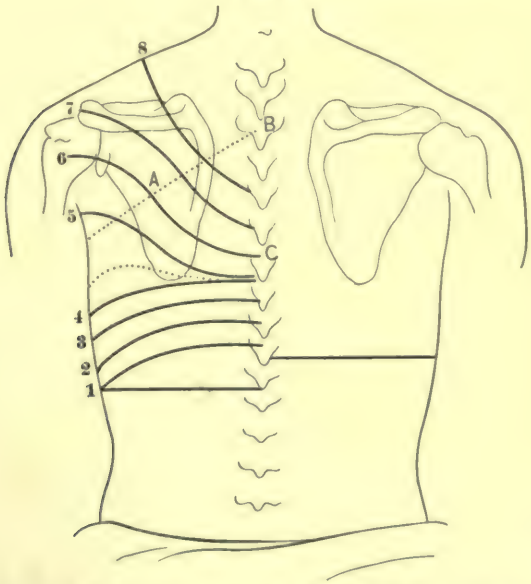
We may best consider the physical signs of effusion as presented in a typical uncomplicated case without old adhesions or any marked disease of the lung. Furthermore, it is both convenient for purposes of description, and also corresponds to real and important anatomical differences, to divide effusions according to their size into small, moderate, and large. A small effusion appears only at the posterior base; a moderate effusion occupies approximately the lower half of the chest, front and back; while in a large effusion the pleural cavity is nearly or quite full, and the fluid exerts a positive pressure in all directions.

1. *Small Effusion.*—The first evidence of the presence of fluid in the pleural cavity is a slight degree of dulness at the extreme base of the chest behind. This zone of dulness may be but an inch or two in width, and extends laterally from the spine for a varying distance toward the axilla. It is often misinterpreted or overlooked, and is recognized only by those who habitually define by careful percussion the lower margin of the lung on both sides behind. The position of these lower margins varies considerably in different individuals according as the lungs are absolutely normal or, as is so often the case, more or less emphysematous; and it is only by comparing the two sides—whereby it is to be remembered that the left is normally a little lower than the right—that a slight unilateral dulness will be readily noticed. Over this dull area respiration is slightly diminished, and immediately above it friction may still be heard. Some German authorities believe that fluid may be detected even before the appearance of this dulness. Weil says that, owing to the presence of fluid in the complementary space, the normal respiratory excursion of the lung into this space is found to be absent. Gerhardt[1] thinks he has often discovered fluid by causing the patient to assume the knee-elbow posture, with moderate inclination of the body toward the affected side; the fluid, he thinks, then gravitates to the axillary region, in the lower part of which it causes a perceptible dulness: it is, however, generally believed that a small effusion is immovable.

[1] *Berliner klin. Woch.*, 1886.

As the fluid increases in quantity these basal signs become more pronounced. The dulness soon changes to absolute flatness, which can be easily and sharply defined on percussion. This flat area has a characteristic and, according to my experience, invariable shape. Occupying the right angle between the spine and the horizontal base of the lung, it is bounded above by a curved line with upward convexity, which, extending outward from the spine, drops, at first gradually and then more abruptly, so as to reach the base at a point near the posterior axillary line. This curve, which I have named the "convex curve" to distinguish it from others hereafter to be described, is shown by the lines 1, 2, 3, and 4 in Fig. 19, which represent varying quantities of

FIG. 19.



Lines 1, 2, 3, and 4 represent the "convex curve" of pleuritic effusion at various stages; lines 5, 6, and 7, the "letter-of-S" curve; and line 8, the "concave" curve. The dotted line above line 4 shows beginning change from "convex" to "S." *A B* represents interlobar fissure. *A B C* is the "dull triangle" of Garland.

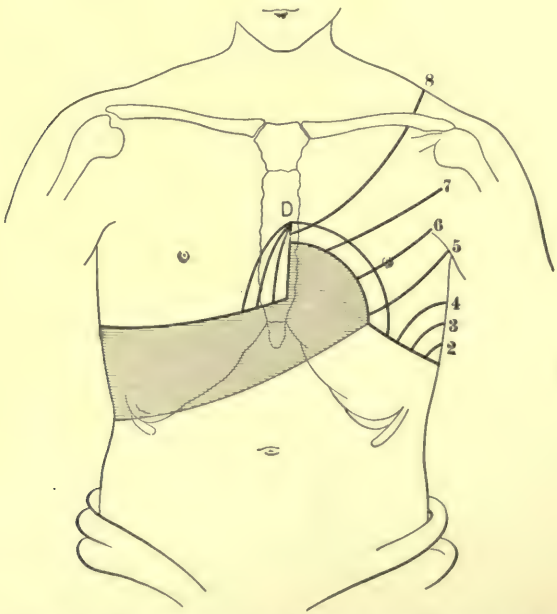
fluid. At first the zone is narrow, not more than two or three inches in width, and the drop is early. As the effusion increases the zone broadens and the drop is farther forward, sometimes reaching the middle or even the anterior axillary line. I have never been able to detect any change in this area on change of position.

Over this zone of flatness vocal fremitus is much diminished or absent. Respiration is usually still heard, though also much diminished in intensity, and is sometimes accompanied by fine moist râles which undoubtedly originate in the lung. I have also heard friction immediately over the flat area. The voice sounds are slightly distant and diffused; bronchophony or ægophony and bronchial respiration are heard over an effusion of this size only when it is complicated by pulmonary consolidation.

Above this effusion respiration is perhaps slightly diminished for a moderate distance, but otherwise there is little if any departure from normal conditions.

2. *Moderate Effusion.*—When a growing effusion has reached a height behind of five or six inches from the base any further increase is forward and upward into the axilla: at the spine it makes a temporary halt. We find, therefore, in moderate effusion a peculiar and characteristic curve of flatness, which was first described by Calvin Ellis, and is now commonly known as the "letter-of-S" curve. Beginning at the spine some five or six inches above the base of the lung, it extends first outward and then sharply upward across the scapula to the upper axilla; from here it may drop abruptly to the base in front near the apex of the heart, or, if the effusion is larger, it may follow approximately the third or fourth rib to the sternum. Frequently it rises so high that it cannot be traced across the axilla, but loses itself in the shoulder. Various S curves are shown by lines 5, 6, and 7 in the fig-

FIG. 20.



Showing "convex" (2, 3, and 4), "letter-of-S" (5, 6, and 7), and "concave" (8) curves, as they appear in front. The different lines dropped from *D* represent varying degrees of cardiac displacement, as found in moderate effusion.

ures (Fig. 19, 20). The S character of the curve is more pronounced in an effusion of medium size; as the quantity of fluid grows still larger the curve straightens and approaches more closely the "concave" curve of larger effusion. This S curve is the only one I have ever encountered in uncomplicated effusions of moderate size, whether recent or of long duration. It is rarely possible to follow carefully the change

from the "convex" to the "letter-S" curve, owing to the rapidity with which it takes place: on one day the former may be found, on the following the latter. I have, however, recently encountered a case where the effusion remained for several days at a point midway between these two curves: the beginning axillary rise of the fluid, as observed in this case, is represented in Fig. 19, p. 269, by the dotted line between lines 4 and 5.

The percussion of the S curve — which I should not be disposed to consider difficult had not so many observers, chiefly foreign, wholly denied that such a curve exists—may be facilitated by attention to the following points: Percussion should be light and should be on straight lines measurably perpendicular to the curve; sharp distinction should be made between dulness and flatness, since the curve represents the dividing line between the absolute flatness of the fluid and the dulness of the lung above; finally, it is sometimes advisable to let the patient take a few deep inspirations in order to facilitate the percussion of the space *A*, *B*, *C* of Fig. 19, p. 269, which Garland has named the "dull triangle:" personally, I have never found such deep inspiration necessary, and it is not to be presumed, because this suggestion is made, that this space is ever devoid of a fair degree of resonance.

Percussion over the retracted lung in moderate effusion presents behind nothing characteristic except the dulness above referred to. In front, however, especially just beneath the clavicle, the note is often exquisitely tympanitic, though at the same time somewhat duller than upon the opposite side. This is probably due to the moderate relaxation of the pulmonary alveoli, which are normally in a state of too high tension for the production of a tympanitic note. This "Skodaic" resonance is often confounded with the tympany hereafter to be described as sometimes heard in very large effusion over the totally compressed lung.

In most effusions presenting the letter-of-S curve intra-thoracic pressure is probably still negative and pulmonary retraction incomplete. But we have seen, in considering the pathology of this disease, that displacement of both mediastinum and diaphragm must begin with the first entrance of fluid into the pleural cavity. This displacement is, however, so slight that even in an effusion of moderate size peculiarly favorable conditions are necessary for its clinical detection. Now, in left-sided effusions such conditions are presented in respect to the heart. The right border of the adult[1] præcordia is normally a perpendicular line extending from the fourth to the sixth rib slightly within the left margin of the sternum, and forming a right angle with the upper border of hepatic flatness. In all healthy individuals above the age of sixteen to eighteen the position of this line is constant and invariable. The lower half of the sternum is therefore normally resonant; that is to say, resonance here is as good as over its upper half, although everywhere over the sternum the note is a trifle high pitched as compared with that of the adjacent lung. Now, this resonant cardio-hepatic angle presents

[1] In children from the age of eight to sixteen the præcordia extends normally beyond the right sternal margin: the writer has called attention to this fact in a paper entitled "The Normal Præcordia in Infancy and Childhood," read before the Pan-American Congress in 1893.

peculiarly favorable conditions for accurate percussion: there is here no disturbing area of relative dulness, but the absolute flatness of the heart changes abruptly to the full tone of the sternum. Hence any encroachment of præcordial dulness upon this normally resonant area is both easily detected and also, in suspected effusion, a measurably sure indication of displacement of the heart toward the right. The different lines dropped from the point *D* (in Fig. 20) illustrate various degrees of cardiac displacement. This is a comparatively early sign of left-sided effusion: we have encountered it even when the anterior limit of flatness did not reach beyond the middle axillary line.

The auscultatory and other signs of moderate effusion, though inferior to those afforded by percussion, are of great value and significance. On inspection the respiratory movement of the affected side is seen to be diminished. Mensuration may show that its circumference is somewhat increased—is not, however, of much diagnostic value. There is no perceptible bulging of the intercostals. Vocal fremitus is usually absent over the flat area, measurably increased over the retracted lung. The respiratory murmur is very much diminished or absent over the fluid; above it is also somewhat diminished in intensity, and near the fluid it is of a quality which has been called indeterminate, being neither distinctly vesicular nor bronchial. The voice sounds over the effusion are distant and diffused, though plainly heard throughout its whole extent; over the retracted lung they are not far from normal, and the abrupt change at the line of flatness is very characteristic. Ægophony is rarely met with in moderate effusion.

3. *Large Effusion.*—An effusion may be so great that pulmonary resonance is entirely obliterated and the whole side is flat to the very summit. Oftener faint resonance is still perceptible over the compressed lung, and the line of flatness can still be defined. The usual statement that this line becomes more nearly horizontal as the chest fills does not accord with my observation. Theoretically, also, it is difficult to conceive how this could be possible, since the lung, unless adherent, does not recede upward into the apex of the pleural cavity, but inward toward the spine. This anatomical fact corresponds, I think, with the clinical, and I have always found the line of flatness in large effusion to be a curve with upward concavity, as represented by line 8 of the figure (Fig. 19, p. 269): leaving the spine at perhaps the fourth to the sixth dorsal vertebra, it passes outward and upward over the shoulder, so as to reach the sternum at about the third rib. Occasionally in front the percussion note within this curve is tympanitic—the so-called "Williams's tracheal tone." This tympany has no relation with the Skodaic resonance heard over a retracted lung. It is not intense, is elicited only by strong percussion, changes its pitch when the mouth is open, and originates in the trachea or large bronchi; rarely it may be accompanied by a metallic or "cracked-pot" resonance.

The most characteristic feature of a large effusion is the displacement of organs, particularly that of the heart and mediastinum, which can always be easily and accurately determined by percussion. The mediastinum appears as a perpendicular line at either sternal margin or even a half inch beyond, and extending from the first to the fourth costal cartilage; on one side of this line is dulness or flatness, on the other

the unimpaired resonance of the well lung. The heart in right-sided effusion is simply displaced, with the mediastinum an inch or two toward the left, as shown by percussion and by the position of the apex beat; at the same time it is slightly tilted upward, so that its apex lies in the fifth, or even in the fourth, intercostal space. In effusion upon the left this displacement is more pronounced. In all typical cases the præcordia is found to the right of the median line—of precisely the same shape as the normal præcordia, though somewhat narrower from above downward. It is bounded above by a curved line which, beginning at the junction of the fourth right costal cartilage and the sternum, passes outward and downward so as to reach the base of the lung near the right nipple. This area, however, is not absolutely flat, nor does it always extend so far to the right: there is usually an outer zone at least of relative dulness, and when the effusion is not excessive the drop of the curve is nearer the sternum. The sounds of the heart when it is thus displaced are heard loudest over the lower sternum near the ensiform cartilage. Its pulsation, too, is often most prominent in the epigastrium, though usually also perceptible, both to sight and touch, at the fourth or fifth interspace in the right parasternal line. Depression of the diaphragm is not, as a rule, so plainly evident. On the right it can only be inferred from the position of the lower margin of the liver, which may, however, in extreme cases be as low as the level of the umbilicus. On the left any considerable degree of depression of the diaphragm can, as a rule, be easily detected. Here there exists, normally, what Traube called the semilunar space—a zone, namely, of well marked tympanitic resonance over the free border of the ribs. This zone is four or five inches in width and roughly semicircular in shape; it is bounded below by the costal margin, above and without by the left lobe of the liver, the border of the lung, and the spleen. Any marked depression of the diaphragm by fluid will cause a perceptible diminution in the width of this zone. Not only may it become entirely obliterated, but in extreme cases the diaphragm may sag below the ribs as a prominent tumor. With any considerable displacement of the diaphragm the spleen is also depressed, and its long axis becomes more vertical. These signs connected with the diaphragm are, as a rule, available only in very large effusions when other diagnostic features are pronounced; except, therefore, as an early indication of absorption, we cannot agree with those observers (Fräntzel, Strümpell, and others) who consider them of very great value.

Inspection in a case of large effusion shows striking changes. The affected side appears markedly distended—much more, in fact, than measurement shows to be actually the case. Both nipple and scapula are farther from the median line than upon the opposite side, and the intercostal spaces are either obliterated or perhaps slightly bulge. The respiratory movements of the opposite lung are usually increased both in frequency and amplitude, while the affected side appears nearly or quite immobile. The pulsation of the heart is noticed in the epigastrium, or near the right nipple, or, in right-sided effusion, considerably outside the left mammary line. Mensuration shows an enlargement of the affected side, which, however, rarely amounts to more than 3 or 4 cm.

Palpation over the effusion discovers an increased resistance in the intercostal spaces as compared with the well side. It also assists in determining the position of the heart's apex. It confirms the evidence of percussion as regards the position of the liver and spleen or any downward bulging of the diaphragm; in rare cases, usually purulent, the latter may be felt below the margin of the ribs as a fluctuating tumor, beneath which, on the right, the whole liver can be freely palpated. Vocal fremitus is, in general, absent over the fluid and exaggerated over the compressed lung; but to this rule there are exceptions, and it may be either everywhere felt, or absent even over the lung itself: these differences are thought to be due sometimes perhaps to adhesions, usually to variations in the perviousness of the bronchi.

The auscultatory signs of a large effusion vary greatly with the degree of pulmonary compression. In an extreme case, when the lung is nowhere in apposition with the chest wall and compression is so great that the lumina of even the main bronchi are obliterated, there is an absolute silence over the whole affected side: the voice is extremely faint and respiration is everywhere totally absent. More commonly the lung is not wholly compressed: it still touches the chest wall within the "concave" curve above described, and its main bronchi, at least, are still pervious. Under these conditions respiration is nearly or quite absent over the fluid, bronchial over the compressed lung. It is not, however, infrequent to meet with a very important exception to this rule. A distinct bronchial respiration may be heard over the whole side from apex to base—an occurrence particularly common in children, but frequent enough in adults as well, and of which there is no satisfactory explanation. It is also to be mentioned that in rare instances of large effusion the respiration may have an amphoric character over the upper and inner front. The voice sounds correspond in the main with those of respiration: when the latter is bronchial there is also bronchophony. But since vocal vibrations are transmitted to a greater distance than those of respiration, bronchophony is usually heard also, though more distant, over at least the upper parts of the fluid. Occasionally, too, the voice may here be ægophonous, a variety of bronchophony of peculiarly high-pitched and plaintive tone, and described by Laennec as resembling a distant echo of the voice itself. Ægophony is not, however, as supposed by Laennec, a distinctive sign of fluid; it is sometimes found in consolidation, and exists in only a small minority of cases of large effusion.

Atypical Forms.—Having now considered the physical signs usually found in typical uncomplicated effusions of varying size, we must allude briefly to those of certain resulting conditions and atypical forms.

Resorption, or the artificial removal of a portion of the fluid, is usually first indicated by a moderate drop in the line of flatness, and in some instances by a perceptible diminution of intercostal tension and a rise of the diaphragm. Voice, respiration, and fremitus also show various slight modifications tending toward a return to normal conditions. All these changes are, however, much less accentuated than might be anticipated, and it is especially to be noted that a return of the heart to its normal position is usually slow and retarded; occasion-

ally it occurs with a perceptible jerk, which may cause a temporary sensation of oppression and great anxiety. A daily measurement of the whole circumference of the chest is claimed by Woillez to furnish valuable evidence as to the progress of absorption. With the disappearance of the effusion there is usually a return of friction in an even more intense degree, and over a larger area, than at the onset of the disease. The numerous crackling râles so commonly heard over the lung during the progress of expansion are ascribed by Traube to the forcible entrance of air into those groups of vesicles which are constantly being opened up as absorption proceeds.

Fibroid Thickening.—The signs of fibroid thickening of the pleura, with consequent retraction of the chest, have several times been alluded to. In addition to the marked evidences of retraction and diminished circumference of the chest to be observed on inspection and mensuration, the percussion note over the lower portion, sometimes the whole, of the affected side is nearly or quite flat. This flat area is usually poorly defined above, and shades off gradually into a fair pulmonary resonance. Respiration below is very faint, and the vocal sounds diminished; above, they vary according to the more or less healthy condition of the lung. Fremitus, on the contrary, is but little if at all affected by pleural thickening. The eventual development of a certain degree of these several signs is, perhaps, the rule after all cases of moderate or large effusion.

Encapsulated effusions have, of course, no typical curve, and produce, as a rule, no marked or typical displacements. Other signs are the same as those of ordinary effusion, except that they are apt to be less pronounced. In those multilocular effusions which develop in cases of old costo-pulmonary adhesions fremitus is said to be often plainly perceptible. Again, adhesions may prevent the usual retraction of the lung toward its hilus, and it may then be found at the base or in a lateral region of the chest: here, of course, percussion outlines may present curious modifications which will suggest themselves to any one familiar with typical conditions.

The *mobility of pleural effusion* on change of posture is still under discussion. Without citing the numerous authorities to be found on either side, it will be sufficient here to make the following statement as one upon which there is very general agreement: Very large effusions are immovable; moderate effusions, reaching perhaps the third or fourth rib in front, do often show change of level on change of posture, but such change is slight, and rarely exceeds the width of one rib and intercostal space; finally, even this slight change of level requires, as a rule, some little time for its accomplishment.

Purulent Pleurisy; Empyema.

Etiology.—If it may be said that the investigations of the past few years have greatly changed our notions as to the origin of serofibrinous pleurisy, this may with still greater truth be affirmed of empyema. Since the first bacteriological studies of purulent effusions, made some ten years ago by Fränkel, Weichselbaum, and others, the number of investigators in this field has been large, and our knowledge

of this affection, though still incomplete, has wonderfully increased. Among the many who have made valuable contributions to this subject may be mentioned Moutard-Martin, Courtois-Suffit, and Netter in France; Ehrlich, Levy, and L. Ferdinand in Germany; and more recently Koplik and Prudden in our own country.

The laborious researches of these and a host of other observers have made it certain that the essential difference between a sero-fibrinous and a purulent effusion is the presence in the latter of certain micro-organisms in considerable quantity. Furthermore, it appears that while various germs are capable of producing pleural suppuration, by far the greater number of empyemas are caused by two or three particular forms, and, finally, it seems that these different germs give rise to forms of the disease which vary greatly in clinical course and outcome. Recent French writers have even ventured to describe these different forms as quite distinct clinical and pathological entities. While we regard it as still a matter of some doubt whether universal experience will eventually sanction these broad distinctions, yet it must be admitted that many facts point in this direction, so that, notwithstanding the as yet somewhat meagre testimony, we feel justified in treating the subject in a measure from this point of view.

Possibly an occasional case of empyema is primary, as will presently appear: the mode of entrance of the germ into the pleural cavity might hypothetically be either from the bloodvessels or through the alveolar walls. Certainly, in the very great majority of cases the disease is secondary to other affections. We may, with a view to clinical convenience rather than on a strictly etiological basis, consider empyema in groups as produced by the following micro-organisms:

1. *Streptococcus Pyogenes.*—As the streptococcus is by far the most frequent germ associated with other suppurations of serous cavities, so it also is with empyema in adult life. For example: Ferdinand[1] in 12 cases found the streptococcus 5 times = 41 per cent.; Prudden[2] in 24 cases 7 times = 29 per cent.; Netter in 156 cases 62 times = 39 per cent.: all of the above represented either pure cultures of the streptococcus in the empyema pus, or at least a great preponderance over other forms. Since, however, many of these cases were children in whom the proportion of streptococcus empyemas is much smaller than in adults, these percentages are far below the average. Thus, Netter found 56 streptococcus cases among 127 in adults alone, or 44 per cent. Probably 40 per cent. would not be too high an estimate of the average frequency of this form in adults. It occurs only about half as often in children.

The sources of streptococcus invasion of the pleura are—(1) The lung: here the pneumonia associated with influenza stands foremost in importance; with other pneumonias the pneumococcus variety is much more frequent. Less common sources are destructive and ulcerative processes, such as tuberculosis, abscess, gangrene, infarction, bronchiectasis, and cancer. (2) Parietal affections: the various phlegmons of the skin, lymphatic glands, and breasts, especially cancer of the breast; also peripleuritis. (3) The mediastinum: cancer of the œsophagus, mediastinal abscesses, and pericarditis. (4) Abdominal suppurations: particularly puerperal peritonitis; also local abscesses, especially the

[1] *Deutsches Arch. f. klin. Med.*, 1892. [2] *New York Med. Journ.*, 1893.

hepatic, subphrenic, and perityphlitic. (5) General diseases of infectious character: puerperal fever, erysipelas, influenza, scarlet fever, and diphtheria.

That empyema is not oftener consequent upon these conditions is explained by the apparent necessity, in order to the development of suppuration, that the streptococci should invade the pleura in considerable numbers, and particularly that there should be some focus, close to or communicating with the pleura, where conditions are favorable for their multiplication. The injection of small quantities of a pure culture into the healthy pleural cavity has usually proved innocuous; cases have also been reported where the presence of streptococci in a sero-fibrinous exudate has evidently shown no tendency to transform it into pus.[1]

2. *The Pneumococcus of Fränkel.*—This organism has been found by all observers in a very large proportion of those empyemas so often consecutive to croupous pneumonia. The same is true of many empyemas following broncho-pneumonia, which is also, as shown by Weichselbaum, frequently produced by the pneumococcus. Again, this germ has been found in a very considerable number of cases apparently primary, where at least there has been no evidence of an antecedent affection of the lung: these cases have the same clinical course as the manifestly metapneumonic, and, while some hold that an unrecognized or latent pneumonia has always preceded, Netter believes that they are often genuinely primary forms.

This variety is very frequent. It may occur at any age, but is exceedingly common during the first decennium, while after the fifth it is rare. Its relative frequency is much greater in childhood than in adult life, as is evident from the following figures: Prudden in 24 cases of empyema at various ages found the pneumococcus in 9, or 37 per cent.; L. Ferdinand in 12 cases 4 times, or 33 per cent.; Netter in 92 cases in adults alone 32 times, or 34 per cent. On the other hand, in children alone Koplik[2] in 12 cases found the pneumococcus in 7, or 58 per cent.; Netter in 29 cases 21 times, or 72 per cent. It would appear, then, from these and other statistics, that in adults the pneumococcus is responsible for about 25 per cent. of all empyemas, and in children for more than double as many, probably at least 60 per cent. Usually, the pneumococcus is found in pure culture; occasionally it is associated with other forms, oftenest the streptococcus or staphylococcus, as occurred, for example, in 20 per cent. of Netter's cases. Many of the latter are to be regarded as secondary infections.

3. *The Bacillus Tuberculosis.*—It is not yet definitely known whether the mere presence of the tubercle bacillus in the pleural cavity can produce an acute empyema. Probably most cases of tubercular empyema are chronic, and the result of a slowly developing tuberculosis of the pleural membrane itself; while the great majority of acute empyemas which occur in connection with pulmonary tuberculosis are non-tubercular, and produced by the streptococcus or other pyogenic organism. Contrary to a very general opinion, tubercular empyema is not a common form of the disease; a sero-fibrinous pleurisy is much more likely to be associated with tuberculosis than an empyema. Netter in

[1] Goldschneider, *Zeitschf. f. klin. Med.*, 1893. [2] *Trans. Am. Ped. Soc.*, 1891.

156 cases encountered only 15 which proved to be tubercular; Ehrlich,[1] however, found 7 cases in 19; Prudden only 1 in 24. Probably Netter's estimate of 10 per cent. as the average frequency of tubercular empyema may be accepted as the most reliable one we have at the present time. This form may appear in the course of florid phthisis, or it may slowly develop in individuals who are apparently in the most vigorous health. The Koch bacillus is found in only a small proportion of tubercular empyemas—by Netter, for example, in only 5 of 13 cases examined, and then only two or three in a field. Inoculation, however, gave positive results in all cases—12 in number—which Netter thus examined. Aside from the tubercle bacillus, a tubercular empyema is likely to contain no bacteria at all, or, at most, the staphylococcus pyogenes.

4. *Other micro-organisms* are exceptional as determining factors in the production of suppuration. The staphylococcus is not infrequently encountered, but nearly always associated with some other species, and apparently playing a subordinate rôle. For example, it appears often in connection with the tubercle bacillus and the pneumococcus. As sole variety it was found by Netter in only 6 of his 156 cases. These pure forms of staphylococcus empyema appear to be usually consecutive either to pyæmic conditions, or to penetrating wounds of the pleura, especially when complicated by the retention of a foreign body. As very exceptional causes of empyema are to be mentioned the bacillus of Eberth and the encapsulated bacillus of Friedländer.

5. *Saprophytic Germs.*—The presence of these organisms is the determining cause of the occasionally fetid or putrid character of an empyema. They have been found in all such cases in much the same variety as they normally exist in the buccal cavity and the intestinal tract. Netter has even succeeded in producing a fetid form of pleurisy by the injection of saliva into the pleural cavity. The invasion of these germs may either provoke an empyema which is putrid from the start or a putrid metamorphosis of an already existing sero-fibrinous or purulent effusion. Their mode of access to the pleura is usually through (*a*) the bronchi: in the great majority of such cases a pulmonary gangrene is the antecedent condition; not necessarily the classical disease, but far oftener minute necrotic foci, such as are often found in the periphery of broncho-pneumonic consolidations. Less frequently the form is secondary to a pulmonary cavity, bronchiectasis, hydatid cyst, or cancer. (*b*) The gastro-intestinal tract: here may be mentioned suppurative anginas or retro-pharyngeal abscesses which penetrate by way of the mediastinum, cancer and stricture of the œsophagus, and the various forms of subphrenic abscess which communicate with the stomach and intestines. (*c*) An external wound: this must have been formerly much more frequent than at present when all wounds and operations are aseptically managed. Putrid infection from without is now exceedingly rare, and is almost limited to old cases of tubercular fistulæ.

Such are the numerous etiological factors of empyema as they appear in the light of recent investigation. They are not always easily discoverable, even on post-mortem examination. It is evident, however, that the disease is finally acquiring a solid etiological basis, and it will pres-

[1] *Berliner klin. Woch.*, 1887.

ently appear that much has been gained with respect to both prognosis and treatment.

Pathological Anatomy.—It is unnecessary here to describe the various conditions, already enumerated, to which empyema may be secondary. Also for the phenomena of displacement, which are in the main identical whether the effusion is serum or pus, reference is made to the pathology of sero-fibrinous pleurisy. In empyema there is somewhat greater tendency to bulging of the intercostals and diaphragm, because pus is heavier than serum, and also because in empyema the surrounding tissues become softer and more yielding. Attention should also be called to the frequent association with chronic empyema of amyloid degeneration of the spleen, liver, and kidneys—a condition which may exceptionally be encountered only a few months from the beginning of pleural suppuration. We may, then, proceed to describe the characteristics of the pus and the condition of the pleura peculiar to empyema.

The exudate is not always frankly purulent in its gross appearances; indeed, there is no sharp line of distinction, even microscopically, between a sero-fibrinous and a purulent exudate, since it is simply a question of quantity of leucocytes. The fluid may therefore present all possible variations in consistency. It may be thin and but slightly opaque, or it may be a thick and viscid pus, bonum et laudabile. The former, on standing, deposits only a shallow layer of leucocytes at the bottom of the glass, with a large quantity of supernatant, often transparent, serum above. On the other hand, the pus may be so thick that no separation into layers occurs. Flocculi or large masses of fibrin and inspissated pus are usually found swimming in the fluid or deposited on the walls and floor of the cavity. The color of the pus is usually yellowish or greenish white or gray; in fetid cases it may present various shades of brown. There is no odor, except in fetid cases, which may be so horribly offensive that the whole room is at once infested. Microscopically, are found a varying number of leucocytes, often also red corpuscles, large granular cells, fat globules and detritus, and cholesterine crystals. Charcot's crystals (see illustration on p. 171) were recently found by Grawitz in a single case; he suggests their possible association with hydatids, in which such crystals are known to occur. It is hardly necessary to again enumerate the various micro-organisms already considered in their etiological relations.

The pathological changes in the pleura and surrounding tissues peculiar to empyema are few. At the outset the process in the pleura itself probably differs but little from that of sero-fibrinous pleurisy. Later there is more extensive and deeper infiltration of the pleura with leucocytes, and its lymphatics are often found engorged with pus. The whole pleural cavity may be involved, or the empyema may be encapsulated between lung and chest wall, or in forms hereafter to be described as diaphragmatic, interlobar, and mediastinal. The walls of the cavity are lined usually with a thick false membrane of yellowish white or grayish color and soft, friable consistency. Sometimes, in cases of long standing, the whole wall is a single granulating surface, the so-called pyogenic membrane. In still other cases the pleural surface seems to be but little altered in gross appearance, if we may trust an

observation to that effect by Laache: after resection in a fresh case of empyema Laache was able, by means of a mirror, to thoroughly inspect the whole interior of the cavity, and found its walls glistening and apparently but little changed. In cases of some standing it is not uncommon to find upon the visceral or parietal pleura spots of erosion, local necrosis of the pleural membrane, so that the underlying lung or other tissue is left bare. These are undoubtedly the first steps in the process of spontaneous evacuation. When such evacuation has occurred through the lung we may find either a rupture of considerable size or no apparent breach of continuity, the pus having filtered through numerous small openings as through a sieve: under such circumstances pneumo-thorax may or may not have supervened. In putrid cases this corroding action of the pus may cause not simply necrosis of the pleura, but also widespread destruction of the pulmonary parenchyma, so that but little of the lung remains except the more resisting bronchi. Parietal erosion may result in an external opening, the pus finding its way through an intercostal space into the subcutaneous cellular tissue, and forming first an external tumor which eventually ruptures; or it may penetrate by other fistulous tracts to more or less distant localities, such as the lumbar region, the groin, the perineum, or the perinephritic region, in which case an abscess may develop occupying half the abdominal cavity and pointing perhaps at the umbilicus. Rarely, also, an empyema may have ruptured into the œsophagus, the pericardium, the opposite pleura, or the pelvis of the kidney. Most evacuations are directly through an intercostal space or into the lung.

Here, as in other forms of pleurisy, there may develop in time great fibroid thickening of the visceral and parietal pleura, with permanent retraction of the lung and thoracic deformity. These thickened pleuræ often contain tubercular and cheesy nodules. Empyema pus may become inspissated, and finally calcified; pleuroliths have been found as large as an orange. Helferich[1] has recently called attention to a new growth of bone which sometimes takes place upon the inner surface of the ribs, so that they appear triangular instead of oval on transverse section.

Symptoms and Course.—Empyema has no typical clinical course. Like sero-fibrinous pleurisy, it may present all possible variations between acuteness and chronicity, between violence of onset and almost absolute latency. At one extreme we have the complex of symptoms which Fräntzel has termed "pleuritis acutissima." Beginning with a severe rigor, there is a rapid rise of temperature to 104° or 105° F., severe pain in the side, and intense dyspnœa. The exudation, which is purulent from the outset, appears early, and rapidly becomes voluminous: it is often putrid. The general condition is markedly affected from the start: there are great weakness and depression, rapid emaciation, dry tongue and sordes, tendency to delirium, rapid and feeble pulse, and, in short, all the concomitants of the typhoidal state. A fatal result may follow at the end of a week, though such fulminant cases are exceptional. As opposed to this pleuritis acutissima we find, at the other extreme, cases of empyema whose onset is most insidious, and whose course is absolutely afebrile. While, however, a generally latent onset and course are common enough, it is at least very unusual for an

[1] *Arch. f. klin. Chirurgie*, xlviii.

empyema to fail to develop sooner or later a certain degree of hectic fever.

Most empyemas occupy an intermediate position between these extreme forms. Their symptoms are either masked at first by the primary affection or the disease develops suddenly as a typical sero-fibrinous pleurisy, with moderate fever, accompanied by pain and dyspnœa. But as the condition progresses the temperature shows no tendency to fall. On the contrary, at the end of a variable period, perhaps ten days or a fortnight, it gradually assumes the hectic type, often with chilly sensations, more rarely with repeated rigors. This septic condition results in a progressive though sometimes gradual emaciation and loss of strength. The face becomes extremely pallid, and the steadily increasing dyspnœa is often out of proportion to the quantity of fluid. Cough is usually troublesome, of a dry character, and not necessarily the result of any pulmonary complication: in case of rupture into the lung it may, of course, be greatly aggravated, with either sudden evacuation of a considerable quantity of pus or a more protracted purulent expectoration. Pronounced clubbing of the fingers develops in older cases, sometimes even in those of only a few months' duration; so also with œdema of the lower extremities, generally associated with an albuminous urine. Finally, if there be no operative interference or spontaneous evacuation, the patient succumbs to general exhaustion, or to a secondary amyloid disease or to some of the complications hereafter to be described.

Having thus briefly indicated the course of empyema in general, we deem it of practical interest and value to note the main clinical characteristics, though still imperfectly established, of the different bacteriological varieties of the disease. In this, as in the preceding section upon etiology, we follow largely the description of Netter, whose splendid work in this field is worthy of the most cordial and universal recognition.

The streptococcus form is not infrequently primary, or, more strictly speaking, it has not been preceded by any recognizable affection. This was true in 16 of Netter's 56 cases. When thus primary it is apt to begin with a rigor and to show a very high range of temperature from the start. The form above described as pleuritis acutissima is probably in most instances streptococcal, though it is not to be supposed that all primary cases are thus fulminant. The form may exceptionally be wholly latent in onset and course, these differences depending apparently upon a varying virulence of the microbe. In general, however, it seems that streptococcus empyema tends to the production of a high and irregular fever, with general symptoms of the typhoidal type. Plainly secondary forms, such as may attend, for example, puerperal peritonitis, pulmonary gangrene, pyæmia, and the like, do not, of course, present any characteristic symptomatology.

Streptococcus empyema has also certain local features which are more or less distinctive. A circumscribed œdema of the chest wall, varying from a slight pale or rosy puffiness to a pronounced doughy swelling which pits on pressure, is particularly frequent in this form. The axillary glands are often enlarged. Exceptionally it gives rise to metastatic processes, among which cerebral abscess, with resulting par-

alysis and convulsions, appears to be relatively most frequent. Encapsulation of the exudate is not so common as in the pneumococcus form, nor is spontaneous evacuation of the pus through the lung or chest wall. The pus of streptococcus empyema is, in general, of but moderate density. Ordinarily, the fluid obtained on exploratory puncture is quite opaque, of yellowish tinge, and on standing deposits a pulverulent sediment in considerable quantity. Often, however, the fluid is but slightly cloudy, and may even be transparent; an intrapleural sedimentation has taken place, and the needle has entered the supernatant layer of serum. It is evident that in such instances it is quite possible to make an error in diagnosis as to the character of the fluid.

The Pneumococcus Form.—We have already seen that this form may be apparently primary, or may be a sequel of croupous pneumonia, sometimes also of broncho-pneumonia. Undoubtedly the secondary form is far the more common of the two, although the antecedent pneumonia is not always discoverable. Netter in 53 cases of pneumococcus empyema obtained a history of pneumonia in only 19; however, 21 of these cases were in children, in whom slight forms of the disease often pass unnoticed. The primary forms may, it seems, begin with much the same complex of symptoms as a frank pneumonia. There may be rigor, pain in the side, cough, and a steady continua for from six to eight days, after which the constitutional symptoms are less prominent. Such cases have recently been reported by Washbourne:[1] in one of these a post-mortem examination on the sixth day of the disease showed an abundant purulent effusion containing pneumococci without pulmonary lesion. Other primary cases may have no characteristic course. Secondary forms also vary greatly in onset and development. A metapneumonic empyema may precede the pneumonic crisis, or it may not appear for two months after the primary affection: usually there is an interval of apyrexia of two to four weeks' duration between the pneumonia and the consecutive effusion. With the latter the temperature again gradually rises, and soon a daily continua becomes established. Netter states that, as a rule, the afternoon exacerbations vary from 102° to 103° F., and that the morning remissions are not usually pronounced. Exceptionally, fever may be surprisingly slight or even wanting. Renvers[2] reports 2 cases, in 1 of which, after a seven days' interval of complete apyrexia, pus was aspirated on the ninth day; in the second case an empyema developed immediately after the pneumonic crisis, but the temperature nevertheless continued normal for seventeen days. Other symptoms of the pleuritic affection are slight. Pain is often absent, and dyspnœa appears only as the effusion becomes large. Some pallor and a moderate tendency to progressive weakness and emaciation are usually noticeable.

But in spite of the irregularity of its course this form of empyema has quite a characteristic physiognomy. In the first place, œdema of the chest wall is decidedly exceptional; it was present in none of Netter's 53 cases, and the same observer could find but 3 instances reported in literature. And yet, on the other hand, spontaneous evacuation is surprisingly frequent in this form. Netter found that it had occurred

[1] *Brit. Med. Journ.* [2] *Charité Annalen*, 1889.

in 25 per cent. of all reported cases, and in his own series the proportion was as high as 40 per cent.—certainly a very notable fact. This evacuation is oftenest through the lungs, though only exceptionally attended by the production of pneumo-thorax. Less often there is rupture through an intercostal space, and even this event is considerably more common here than in any other variety of empyema. Furthermore, there is noticeable in this form a decided tendency to encapsulation; many of the cases of interlobar, diaphragmatic, and other local empyemas are pneumococcal. Very important and distinctive is the character of the pus. It is, as a rule, of a dense creamy consistency, rich in fibrin, and therefore extremely viscid. Its color is a greenish yellow, the shade of green being usually so pronounced as to make it a special diagnostic feature. The pus is also rich in fibrinous clumps and flocculi. Finally, pneumococcus empyema is of peculiarly benign course and prognosis. Most cases end in complete recovery, and it is unquestionably in this form that so many favorable results have followed the milder methods of operative treatment. Hence also the favorable course of most empyemas in children; and the complete accord between this long-established clinical fact and the more recent evidence of bacteriology is certainly notable.

The *tubercular form* is fully described elsewhere in this work,[1] and need here be but briefly alluded to for purposes of comparison with other varieties. This form may perhaps occasionally be acute; certainly its more usual and characteristic course is chronic, of latent type and insidious development. It may apparently be purulent from the outset, or, what is probably the rule, it begins as a sero-fibrinous pleurisy, the exudate of which repeatedly recurs after withdrawal, and eventually, without general symptoms of infection, acquires a purulent character. Unquestionably, many of the cases in which aspiration has incurred the suspicion of having changed a simple into a purulent effusion belong in this category. The pus in tubercular empyema is usually thin, gravish, pulverulent, poor in leucocytes, and not infrequently chyliform or fatty. Sometimes it perforates the thoracic wall, and appears externally as a cold abscess which in rare instances may pulsate synchronously with the heart. The form is not infrequently complicated with pneumothorax. Its diagnosis may occasionally be made by the discovery of the Koch bacillus; and its presence is always to be considered probable when the pus contains no pathogenic germs or only the staphylococcus. The general condition in tubercular empyema when uncomplicated by advanced phthisis or by the presence also of pyogenic germs may remain good for an indefinite period, and months or even years may elapse before health becomes seriously impaired and the disease advances toward a fatal termination. It is, however, not to be forgotten that both streptococcus and pneumococcus forms may, and do frequently, occur as secondary affections in connection with pulmonary tuberculosis, and that complete recovery from the empyema is then possible.

Fetid and putrid forms of empyema we have already seen to be invariably due to the invasion of the pleural cavity by saprophytic germs. We have also seen that oftenest the source of infection is some form of cortical pulmonary gangrene, which, however, may not neces-

[1] Vide Osler on "Tuberculosis," Vol. I. p. 767.

sarily have opened into the pleura. It is evident, therefore, that a putrid empyema may originate as such—may be *putride d'emblée*, as it is often termed—and Netter believes that the great majority of cases do begin in this way. The onset of the disease is then extremely violent. There is an initial rigor, followed by an immediate rise of temperature to a very high degree. Pain in the side is of extreme and unusual severity. The tongue soon becomes dry and brown, prostration is very marked, and, in a word, the patient rapidly develops a condition of profound sepsis. Other cases, again, are less stormy in origin, particularly those forms which develop out of an originally sero-fibrinous or simply purulent exudate. This is most likely to happen when the effusion has been of long standing, and for this reason, as well as others, tubercular effusions show an especial tendency to this change. Putrid empyema is very often complicated with pneumo-thorax.

Although the symptoms and course of putrid empyema present nothing absolutely characteristic, there are suggestive features. The initial pain is often of unusual severity and persistence. Fever shows a tendency to a high range, and the daily oscillations are likely to be very great. Occasionally a fetid expectoration is met with even when there is no direct communication with the bronchi—a phenomenon analogous to the fecal odor of abdominal abscesses, which are simply contiguous to the large intestine; in case of rupture into the lung there may of course be continuous evacuation of large quantities of fetid pus. The most characteristic feature of putrid empyema is the constitutional condition. As a rule, there is an early and pronounced development of sepsis. The tongue is brown and parched; there are abundant sordes, great weakness and depression, stupor, and marked tendency to delirium. This same condition may, it is true, accompany the streptococcus form, but it is then exceptional, here the rule. The fluid withdrawn on exploratory puncture may be simply fetid or it may have the almost unbearable odor of gangrene. Sometimes such a pus is thick and creamy, but usually it is thin and sanious, its color varying between a grayish yellow and the different shades of brown. On standing it deposits a sediment which is usually powdery and of moderate quantity. This sedimentation may be intramural, and the syringe may therefore, in exceptional cases, aspirate an almost transparent serum. Its microscopical constituents are white and red corpuscles, varied cellular débris, crystals of fat acid and cholesterine, fat globules, and microbes. The putrid character of the pus may disappear in a very few days after operative treatment.

Other forms of empyema have been too little studied and are too infrequent to make any attempt at characterization possible. Those containing solely the typhoid bacillus of Eberth have usually proven benign in course. Pure staphylococcus forms seem to be often destined to become tubercular if life is sufficiently prolonged.

The PHYSICAL SIGNS of empyema are in the main those of sero-fibrinous pleurisy, as described on page 268. The character of the fluid is an absolutely indifferent factor as regards its effect upon auscultation and percussion. Baccelli, it is true, thought he had discovered a differential sign of great value in the non-transmission of the whispered voice through a purulent effusion; but this may also be the case in both serous

and hemorrhagic forms, and exceptions are so frequent that the sign is now generally admitted to be unreliable. Displacement in empyema is, on the whole, apt to be more pronounced than in sero-fibrinous pleurisy, and it is chiefly in the former that instances of enormous downward bulging of the diaphragm, such as to produce prominence of the hypogastrium and a palpable fluctuating tumor, have been observed. Local œdemas and the various qualities of empyema pus have already been sufficiently described.

Special Varieties of Pleurisy.

Diaphragmatic Pleurisy.—An acute inflammation limited to that portion of the pleura which covers the diaphragm and the under surface of the lung may sometimes give rise to a peculiar complex of symptoms. The variety is not common. It may be secondary to contiguous abdominal affections or it may be primary, and, in general, subject to the same causative influences as other pleurisies. The dry form is exceptional; most cases are accompanied by effusion of either sero-fibrinous or, more rarely, purulent character. The onset of the disease is usually brusque. It may begin with a rigor, and, as a rule, the febrile movement is pronounced, frequently 103° to 104° F. The initial pain is extremely severe; it is in most cases referred to the hypochondrium, but may be felt along the costo-phrenic attachments or low down in the back. The face is anxious, the pulse rapid and small, and dyspnœa often excessive. The characteristic physiognomy of this form of pleurisy is, however, chiefly due to the great tenderness of the inflamed diaphragm which the patient tries in every possible way to immobilize. To this end he may sit slightly bent with hands pressed against the sides, or he may take the semi-recumbent posture with elevated knees. Respiration is rapid, costal, superficial. The abdominal wall, especially on the affected side, is tense and its muscles are firmly contracted. The abdomen is extremely sensitive to pressure. Other tender points are also to be found along the course of the phrenic nerve, especially between the attachments of the sterno-cleido-mastoid and in the interspaces along the sternal margin: also over the costo-diaphragmatic attachments, and particularly at a point which lies at the intersection of two lines—one a prolongation of the right sternal border, and the other drawn horizontally at the level of the osseous portion of the tenth rib. These tender points are the result of a slight neuritis of the phrenicus, which may persist even after the pleurisy has disappeared. Pain is also increased by cough, by the hiccough which is occasionally a troublesome symptom, and even by all attempts to speak. Vomiting, though not constant, is a frequent and important symptom: it not only greatly aggravates the pain, but it often proves a very misleading factor in diagnosis. The bowels are usually constipated. In the graver forms delirium is frequent, and is apt to be a precursor of fatal coma. Physical signs characteristic of pleurisy are usually lacking. Owing to the immobility of the diaphragm the respiratory murmur at the base of the chest is deficient; sometimes friction appears in the same region, or, in case of effusion, a zone of flatness with some downward displacement of liver or spleen.

Especial interest attaches to the diagnosis of this form of pleurisy because of its clinical resemblance to peritonitis and to other serious abdominal affections. The violent onset, anxious countenance, rapid pulse, vomiting, abdominal pain and tenderness, and flexed thighs are certainly far more suggestive of intestinal perforation than of any thoracic disease. Of 5 cases observed by Fenwick,[1] 1 was considered an attack of gall-stones, another typhlitis, and a third acute peritonitis. The diagnosis is assuredly not always easy. An important step will have been taken when the possibility of diaphragmatic pleurisy is once considered and a thorough examination made with reference to it. Friction or other local signs may then afford valuable evidence, or the characteristic points of tenderness above referred to may be discovered. It is to be especially noted that in pleurisy the abdomen is not distended; the pain is often superficial, and is more closely related to cough, deep inspiration, and like efforts than in peritonitis; and dyspnœa is a symptom of far greater prominence than in any abdominal affection.

The termination of this form of pleurisy is usually favorable. Fatal cases are either purulent or complications of grave affections like cancer, tuberculosis, or peritonitis. When these cannot be excluded the prognosis must be somewhat guarded.

Hemorrhagic Pleurisy.—It is frequently stated that all sero-fibrinous exudations contain a certain number of red blood corpuscles, though usually less than the 6000 per cubic centimetre found by Dieulafoy to be requisite for the production of the faintest tinge of color. Grawitz,[2] however, seems to have found red corpuscles in only 10 of 48 cases carefully examined; and, since 8 of these 10 cases were tubercular, the question arises whether even a minimum quantity of blood in an effusion has not a certain diagnostic value. But we are dealing here with exudations which are frankly hemorrhagic, which vary in color from a rose tint to deep red or brown, and contain sometimes as high as 10 per cent. of pure blood. Such exudations are not infrequent. At the clinic in Munich, for example, this form was encountered 14 times in 227 cases of pleurisy. As a rule, one of the following conditions is responsible for its occurrence:

(*a*) *Tuberculosis*, probably, in most cases, of the pleura itself. The hemorrhage is a consequence of the great vascularity of the tubercular sero-membranes, and of the marked tendency to degenerate changes in the walls of the newly-formed vessels. A hemorrhagic exudation may accompany all the various forms of tubercular pleurisy. Since the amount of blood is nearly always small, the color of the exudate rarely presents the deeper tones of red observed in other forms. The hemorrhage, as such, has no influence upon the prognosis of the disease. Occurring in chronic forms of pulmonary or pleural tuberculosis, a hemorrhagic exudate generally loses a portion of its color with each succeeding aspiration, and after six or eight punctures the effusion itself often finally disappears. It is hardly necessary to say that the great majority of tubercular pleurisies are, microscopically at least, non-hemorrhagic.

(*b*) *Pleural or Pulmonary Cancer.*—A hemorrhagic effusion may

[1] *Lancet*, 1893. [2] *Loc. cit.*

develop at any period of the cancerous growth. Usually latent and unnoticed, it may exceptionally be of acute and violent onset. Dutil,[1] for example, saw a case which began in an apparently healthy individual with severe pain and dyspnœa; there was no return of the effusion after a single aspiration, but the patient died not long afterward of pulmonary cancer. The admixture of blood is usually considerable; the color is apt, therefore, to be dark red or brown and the quantity of fibrin large. As a rule, the fluid persistently re-forms after withdrawal, and, unlike a tubercular effusion, maintains its hemorrhagic character until death.

(*c*) *Pleural Hæmatoma.*—Whenever a serous membrane is the subject of recurrent inflammation it may involve not only the membrane proper, but also any organized false membrane which lies upon it. The delicate vessels of the latter are easily ruptured, and from them a considerable quantity of blood may escape, either into the serous cavity or into the loose meshes of the membrane itself. Occurring in the pleura, this process may give rise to hemorrhage precisely as a pachymeningitis may produce a hæmatoma of the dura mater. Although it seems probable that most of these cases are tubercular,[2] it is certain that the condition may occur in apparently healthy individuals and end in complete recovery. The quantity of blood is often considerable, but two or three aspirations are sometimes sufficient to effect a permanent cure.

A hemorrhagic exudation from other causes than the above is very infrequent. It may exceptionally be associated with cardiac or renal disease or with cirrhosis of the liver. In persons also affected with any form of the hemorrhagic diathesis, such as scorbutus, hæmophilia, purpura, pernicious anæmia, icterus, or leucæmia, a pleuritic effusion is liable to be hemorrhagic in character.

The diagnosis of this form of pleurisy is rarely possible except by exploratory puncture, whereby it is not to be forgotten that any effusion may be slightly tinged with blood if the lung happens to have been wounded by the needle. In very exceptional cases the hemorrhage may be so considerable as to cause marked pallor and weakness. The proportion of blood in an effusion may be estimated by counting the red disks, or, if these are so pale as to make this method difficult, recourse may be had to Gowers' hæmoglobinometer, as recently recommended by Henry.[3] The treatment of a hemorrhagic effusion does not differ from that of the ordinary sero-fibrinous form.

Chylous Pleurisy.—A pleural effusion resembling chyle is an exceedingly rare occurrence. Boulengier[4] has given careful study to reported cases, and concludes that two forms must be sharply differentiated: In the first place, the pleura may unquestionably contain a genuine chyle, consisting solely of a pure emulsion of fat, and the result of an injury to the thoracic duct, or, possibly, as Boulengier thinks, to the operation of the filiaria sanguinis. Busey[5] has succeeded in collecting only 10 cases of genuine chylo-thorax in literature: in 5 of these the chyle came directly from the thoracic duct, 3 being traumatic. Turney[6] has since observed a case due to obstruction and rup-

[1] *Gaz. méd. de Paris*, 1887.
[2] Mesnil, *Thèse de Paris*, 1894.
[3] *Med. News*, 1888.
[4] *Presse médicale Belge*, 1890.
[5] *Canadian Pract.*, 1891.
[6] *Lancet*, 1893.

ture of the thoracic duct from cancerous thrombosis of the subclavian and jugular veins. Busey considers the diagnosis impossible without puncture, and the prognosis unfavorable. In the second form, or pseudo-chylo-thorax, the simply "chyliform" effusion is a result of the fatty metamorphosis of the cellular elements of an ordinary sero-fibrinous or, more commonly, purulent effusion. This change takes place only in cases of long standing, and a very large proportion of such empyemas are tubercular. The microscope shows numerous fat globules, degenerated leucocytes and epithelium cells, and cholesterine crystals, which are suspended in an albuminous fluid. Boulengier thinks that a sufficient number of cellular elements will always have survived to make a differentiation from the true form possible. These chyliform effusions have the same prognosis and treatment as any chronic empyema.

Pulsating Pleurisy.—Although in any large effusion upon the left a pulsation isochronous with that of the heart may exceptionally be felt or seen in several intercostal spaces of the affected side, most cases of pulsating pleurisy are purulent, and the pulsation is limited to a well-defined tumor produced by an external pointing of the pus. The condition is very unusual. Since attention was called to it in 1844 by McDonnel some 50 cases have been reported. The pulsating tumor is almost invariably on the left front and between the second and sixth ribs: of 42 cases reviewed by Osler it was behind in only 3. Usually there is but one tumor, exceptionally two or even more. The pulsation is often intensified when the patient lies upon the opposite side. Comby finds that the great majority of cases are complicated by pneumo-thorax, and that in certain positions of the patient the tumor may contain nothing but air. There is no very satisfactory explanation of this phenomenon of pulsation, but the fact that it generally ceases with the aspiration of a small quantity of fluid makes it probable, as supposed by Traube and Bouveret, that the necessary conditions are a considerable degree of fluid tension with a locally diminished resistance of the chest wall. The prognosis of these cases was formerly thought to be invariably fatal, and it is certain that very many of them are tubercular. Light,[1] however, has observed at least one complete recovery after a duration of only two months. The treatment of an empyema is in no way influenced by the mere presence of pulsation.

Encapsulated Pleurisy.—Under this term we here include not only effusions limited by costo-pulmonary adhesions, but also those commonly described as interlobar and mediastinal—effusions, namely, which are shut in either between two lobes of the lung or between the lung and mediastinum: the two latter forms are nearly always purulent.

The only peculiar interest which attaches to encapsulated pleurisies concerns their physical signs and diagnosis. A dulness or flatness of greater or less extent is usually produced, but this area has no typical outline. Over it voice, respiration, and fremitus are, it is true, usually diminished, sometimes absent; but the small size of the effusion perhaps, or contiguous areas of compressed or consolidated lung, are apt to produce confusing modifications of these signs. In interlobar effusion the dull area is usually found in the axilla or crossing it in the direction

[1] *Lancet*, 1891.

of the fissure. A mediastinal pleurisy may produce an area of flatness which includes the whole width of the sternum and extends quite a distance beyond toward either side. When on the left it may displace the heart toward the right, and by pressure on the great vessels cause marked cyanosis, dilatation of the superficial veins of the chest, puffiness of the face, cold extremities, and œdema of the upper part of the body.[1] The diagnosis of an encapsulated pleurisy is often impossible without exploratory puncture. One is aided by any local effacement of the intercostals, and especially by an absolute flatness with sense of great resistance on percussion. A mediastinal effusion differs from a pericardial in its atypical curve—from mediastinal abscess sometimes in its greater extent and in production of greater displacement of the heart. In all these forms must be taken into consideration a host of diagnostic factors which cannot here be discussed. The treatment of an encapsulated effusion must be conducted on the same principles as that of the ordinary forms.

Pleurisy at the extremes of life has certain peculiarities. In the aged it is found oftenest with pneumonia, chronic cardiac and renal disease, and cancer. There is rarely much fever or pain, dyspnœa is moderate, and the course of the disease is slow. The outcome is always dubious. In children, except the very young, acute sero-fibrinous pleurisy is an extremely benignant affection. It has an average duration of only one to two weeks, and is rarely followed by any permanent deformity of the chest even when the effusion is large. Latent forms are also frequent, though these also, unless purulent, tend toward spontaneous recovery. The special characteristics of empyema in children have already been duly considered (page 283).

COMPLICATIONS OF PLEURISY.—Most important among these are certain exceptional conditions or accidents which may result in sudden death; also certain peculiar attacks of a very serious nature which occasionally follow aspiration or may attend the after-treatment of empyema.

Sudden Death.—It appears to be at least no very extraordinary occurrence for a large pleuritic effusion to terminate very suddenly and unexpectedly in death. Dieulafoy in 1872 was able to collect 40 reported cases of this kind. These sudden deaths may occur during the first week of a rapidly growing effusion, or much later at a time when the patient is apparently convalescent. The causes of this event are undoubtedly varied. Contrary to what might be anticipated, in about two thirds of the reported cases the effusion was on the right, and the hypothesis of Trousseau, that death usually results from a twisting of the great vessels consequent upon cardiac displacement, has necessarily been abandoned. In a certain proportion of cases the condition is one of syncope, due either to fatty degeneration of the heart or to an undue pressure upon it (Garland; Lichtenheim), or, in left-sided effusion with great displacement, to an acute bending and consequent obstruction of the inferior vena cava (Bartels; Fräntzel). Other cases, constituting, according to Goupil and Talamon, a considerable majority, are caused by a thrombosis of the right heart or pulmonary artery. The patient is then seized with great dyspnœa and epigastric pain; the face becomes

[1] Velimirovitch, *Étude sur de Pleurisie médiast., etc.*, Paris, 1892.

anxious and cyanotic; the heart's action tumultuous; the pulse small and irregular; and in ten or fifteen minutes, at most, a fatal termination follows. Other possibilities of sudden death are from pericarditis, from syncope during the aspiration of fluid, and also from collapse such as may follow those forms of pleural irritation presently to be described.

Pulmonary œdema may appear on the well side in cases of excessive or rapidly developed effusion, or on the affected side when aspiration has been too rapid or too abundant. The probable explanation of the latter is a temporary vaso-motor paralysis of the vessels of the compressed lung, permitting a ready transudation of serum when pressure has been too suddenly and completely removed by aspiration. The prominent symptoms of œdema are sudden oppression and dyspnœa, distressing paroxysms of cough, and particularly an abundant serous expectoration; sometimes this serum is raised in enormous quantities, amounting to even one or two litres in a few hours; and, since it is very rich in albumin, the condition has often been described as "albuminous expectoration." The physical signs, as observed in the previously unaffected lung, are numerous moist râles without much dulness on percussion. Most cases recover after a few hours of very considerable distress and danger. Occasionally expectoration is insufficient, the lungs fill, and the onset is rapidly followed by death. This complication is an infrequent one, especially since effusions are no longer allowed to become extensive and the proper method of aspiration is generally understood.

Reflex complications of the most serious nature have long been known to occasionally follow even the most trifling manipulations involving the pleura. In very rare instances death may immediately result, as, for example, I have known to happen after simple exploratory puncture with a hypodermic needle; such a case, however, could hardly be explained except on the hypothesis of a diseased heart. Of much greater frequency and interest are certain cerebral attacks, associated usually with operated empyemas, and following such apparently trivial procedures as the reinsertion of a drainage tube, probing of the pleural fistula, or a simple non-medicated lavage. Jeanselme[1] has collected 46 cases of this sort, and that such accidents are not infrequent may be inferred from the fact that Laache[2] personally observed 3 cases, 1 of which was fatal. At least two distinct forms may be encountered (Jeanselme; Cerenville)—the convulsive form, sometimes called pleural epilepsy, and the hemiplegic. Several days may elapse between the washing or other manipulation and the attack. Talamon[3] reports a case in which, two days after puncture, there were convulsions lasting six hours, followed three weeks later by a second attack with fatal termination: this person was not epileptic, nor have any of the reported cases been in epileptic individuals. The hemiplegic form is usually, though not invariably, attended with unconsciousness. Dilatation of one pupil and bleeding or great sensitiveness of the fistula are said to be common precursors of an attack. A cerebral embolus may be concerned in the production of a certain number of these cases, but most of them can only be explained as reflex—possibly the cerebral centres, as supposed by Jeanselme, being in a state of increased sensibility from an auto-

[1] *Revue de Méd.*, 1892.
[2] *Deutsche med. Woch.*, 1894.
[3] *La Méd. moderne*, 1893.

intoxication. The prognosis of either form is always extremely grave, the mortality having hitherto been about 50 per cent.

Of other complications of pleurisy, pericarditis and peritonitis are the most important. The former may result from any variety of inflammation involving portions of the pleura contiguous to the heart. It may be attended with effusion, and is not an uncommon cause of death: out of 47 cases of empyema, Laache lost 3 from what he describes as a chronic and insidious form of pericarditis. Peritonitis is certainly an infrequent complication of pleurisy, and its occurrence is almost wholly limited to the purulent forms. It is rapidly fatal when general—may, however, be subphrenic and local. Empyema may also be the source of metastatic abscesses, and these appear to have an especial predilection for the brain; Hadden reports 3 fatal cases following empyema, 1 of which was of the same fetid character as the primary disease. Empyema may also be complicated by an erysipelas starting from the external wound.

Acute pneumonia of the affected side is almost never a direct consequence of pleurisy, though extremely frequent as an antecedent: occurring in the opposite lung, it is very apt to be fatal. Bronchitis, usually mild and unaccompanied by expectoration, is quite common. Chronic interstitial processes in the lung have already been referred to as possible sequelæ of old and recurrent forms, and the relation of pleurisy to the subsequent development of pulmonary tuberculosis need not again be emphasized. Pleurisy of the opposite side is considered by most authorities to be some indication of the tubercular character of both; certainly cases of double pleurisy may make at least a temporary recovery.

Diagnosis of Pleurisy.—The early recognition of an acute pleurisy depends almost wholly on the presence of friction. In its absence mild afebrile cases are hardly to be distinguished from intercostal neuralgia or pleurodynia, while the more severe are liable to be confounded with pneumonia. In this connection it is to be remembered that in pleurisy an initial rigor is rare, dyspnœa is more superficial, and there is no rusty sputum; moreover, the early physical signs of pneumonia are missed. We know of no way in which the dry form of pleurisy can be distinguished from the sero-fibrinous before the development of effusion; in general, the latter is perhaps attended by more pronounced constitutional symptoms. It is always best to suggest the possibility of an eventual collection of fluid.

There are few diseases of which the diagnosis can be made with greater accuracy and certitude than that of a typical pleuritic effusion. Among the physical signs which make this certainty possible we do not hesitate to accord the foremost rank to those afforded by percussion. The shape of the flat area is the only constant and invariable sign of small and moderate effusions, while the existence of a large effusion is at once demonstrated by the evidences of displacement. We do not underestimate the value of other signs. The absence of vocal fremitus is quite constant and characteristic, and when combined with absent or diminished respiration and distant voice it makes an effusion exceedingly probable. But we have seen that all these signs may be wanting. We may hear over an effusion both bronchial respiration and bronchophony,

fremitus may be distinct, and there may even be râles. This might, indeed, be anticipated when we consider the great number and variability of the factors involved in the transmission of sound from the larynx. The only constant signs are an absolutely flat area of definite shape and certain phenomena of displacement.

We believe, therefore, that the diagnosis of uncomplicated non-encapsulated pleuritic effusion, whether sero-fibrinous or purulent, should rest primarily upon the following signs: In small effusion an area of flatness at the posterior base bounded above by the "convex" curve; in moderate effusion an area of flatness which is highest in the axilla, and whose upper boundary behind is the "letter-of-S" curve (see pp. 269, 270, Figs. 19 and 20); in very large effusion a curve with upward concavity which crosses the shoulder; and, still more important, displacement of the heart, mediastinum, and diaphragm.

From pneumonia diagnosis by the aid of these signs is generally easy. In consolidation of a lower lobe, the upper border of dulness is highest at the spine, from which, following the fissure, it descends by a gentle curve to a point on the sixth rib just outside the mammary line. In effusion these conditions behind are reversed: the line of flatness is lowest at the spine, highest in the axilla. A greater difference could hardly exist than that between the dotted line of pneumonia in Fig. 19, page 269, and any of the "S" curves also represented. The value of the signs of displacement in these cases liable to be mistaken for pneumonia is slight, since, as a rule, but little perceptible displacement exists. It is only when on the left that a moderate effusion causes an easily perceptible displacement of the heart toward the right, which is of considerable assistance in the diagnosis between these two affections.

Other signs are not to be neglected. Increased vocal fremitus, loud bronchial respiration and bronchophony, and numerous râles are certainly the rule in consolidation, the exception in effusion. While no one of these signs approaches in value the evidence afforded by percussion, their confirmatory value is inestimable.

Considerable difficulty in diagnosis is often presented by old cases of pleurisy with great thickening of the pleura and possibly also fibroid changes in the contiguous lung. The lower part of the chest is found to be dull or flat, with distant respiration and voice. In distinguishing this condition from effusion the following points are serviceable: In the former there is often marked retraction of the chest wall; there is a history of chronic disease; the upper border of flatness is not always sharply defined, and its curve is often atypical; vocal fremitus is often but slightly diminished or normal; and the heart is frequently drawn toward the affected side, while the opposite lung shows signs of emphysema.

Effusion upon the right may be simulated by an upward displacement of the liver or a subphrenic abscess. Here, again, the line of flatness is the determining factor in diagnosis. In both of these conditions we find, as a rule, a symmetrical elevation of the pulmono-hepatic boundary—a sharply defined horizontal line of flatness at the level of the fifth, fourth, or possibly even the third rib. We have seen that in effusion such a line is never encountered, except possibly in rare instances of the diaphragmatic form. An hepatic abscess or cancer does sometimes cause an irregularity of the upper hepatic boundary,

but it is very questionable whether this could ever closely simulate the curve of effusion.

Atypical or encapsulated effusions, such as result from adhesions which limit the exudate or prevent the ordinary retraction of the lung toward the spine, often present no absolutely characteristic signs. They are liable to be mistaken for pneumonic consolidations, abscesses of the lung, bronchial glands, or mediastinum, peripleuritic abscess, hydatids, and malignant new growths. It is impossible here to specify the numerous points of diagnosis involved. After carefully weighing the history, the age and general condition, the degree of local bulging perhaps, or of displacement, an exploratory puncture will, in most cases, afford the only certain evidence of fluid.

For the recognition of the nature of an exudate we are also compelled in most cases to resort to exploratory puncture. It is precisely in the differential diagnosis between serous, hemorrhagic, and purulent effusions that exploratory puncture finds its widest field of usefulness. A localized œdema is a fairly reliable sign of pus, but is found only in a small minority of cases, and may even accompany the non-purulent forms. The so-called sign of Baccelli—the transmission, namely, of the whispered voice through a serous exudation only—though not devoid of value, is now generally admitted to be unreliable; exceptions are too frequent. A daily hectic is strongly suggestive of pus—may, however be present in sero-fibrinous pleurisy, while in empyema the rise of temperature may exceptionally be very slight. Still, in cases where with daily hectic there are occasional rigors, or great weakness and emaciation with a pale, puffy face and slight œdema of the extremities, the effusion may safely be considered purulent.

Hemorrhagic effusion presents no characteristic physical signs. It can only be occasionally suspected in cases of cancer, pulmonary tuberculosis, or hemorrhagic diathesis, especially when a noticeable pallor has rapidly developed. The diagnosis of air above the fluid must depend upon succussion and the evidence of a changing horizontal level of fluid. Hydro-thorax has the same curve as effusion, but is rarely unilateral, and is usually associated with general anasarca dependent upon chronic affections of the lungs, heart, or kidneys.

Exploratory puncture has been frequently alluded to as often essential to diagnosis. We consider it an absolutely harmless procedure when carefully done, and a legitimate method of diagnosis in all conditions and ages. The best instrument is an ordinary hypodermic syringe, but the needle should be of double length, since the usual short one will seldom reach the fluid. The needle should be boiled before puncture, and the syringe, filled with a 5 per cent. solution of carbolic acid, may be immersed for half an hour in a like solution. The hands and the patient's side should be aseptic. The pain produced is very slight, and may be made still less by the use of cocaine. Puncture may be made at any point within the flat area, preferably not too near the boundary, and at the base of the effusion in order to reach the thickest portion of the fluid. It is not to be forgotten that puncture above may withdraw only cloudy serum, even though the exudation be frankly purulent. In small effusion we puncture low down and a little within the line of the angle of the scapula; in moderate and large, in the sixth

or seventh space in the axilla. Occasionally, when the needle is obstructed and no fluid obtained, sufficient pus may have adhered to the end of the needle to be plainly detected by the microscope. That results of great importance are to be obtained by the various methods of bacteriological examination need not again be emphasized. For the technique of these methods reference must be made to appropriate works.

Prognosis of Pleurisy.—In presence of a pleurisy of whatever form we must first attempt to discover its possible association with other conditions. It is evident that the prognosis of a pleurisy which accompanies puerperal or malignant scarlet fever or hepatic abscess depends but little on the pleurisy itself. So also with renal and cardiac affections and advanced pulmonary tuberculosis: the complicating pleurisy is often important only as a terminal affection. We can only consider here the probable outcome of such cases of primary pleurisy as are apparently uncomplicated by any grave general condition.

Simple dry pleurisy is almost never serious, although the possibility of the eventual development of fibroid lung or tuberculosis must be remembered. Smith[1] reports that of 16 cases of uncomplicated dry pleurisy under constant observation during fourteen years, 5 developed some form of tuberculosis.

The immediate prognosis of primary sero-fibrinous pleurisy is favorable. Engster[2] reports 3 deaths in 107 cases; Catrin,[3] only 1 death in 75. The mortality is greatest at the extremes of life, and there is always some immediate danger when the effusion is large and attended by marked displacement, or, particularly, when the case is complicated by pericarditis. A small or moderate effusion may, in general, be expected to recover fully in from four to eight weeks. Rheumatic cases are said to be especially benign and of only a few days' duration.

The remote prognosis is, however, always uncertain. We have already seen with what frequency pulmonary tuberculosis follows even the mildest cases of primary pleurisy. More or less unfavorable symptoms are high and persistent fever, very large amount of fluid, no signs of absorption after the third or fourth week, and rapid recurrence of the exudate after aspiration. Under these conditions especially, and also in any of the chronic latent forms, the tubercular nature of the affection and the subsequent development of other more serious forms of tuberculosis must be regarded as highly probable. Still, even here there may be ultimate complete recovery, or the effusion may be carried about for an indefinite period with maintenance of fairly good health: such cases of chronic effusion have lived twelve or fifteen years. After large effusions and all chronic forms there is always, except in childhood, a probability of some permanent deformity of the chest.

The outcome of empyema when left to itself is usually fatal. Very exceptional cases may recover by spontaneous evacuation, or, still more rarely, a purulent exudate may undergo fatty metamorphosis and be absorbed. As a rule, if the patient escapes the various complications from pressure, inflammation by contiguity, and metastasis, he succumbs eventually to sepsis and progressive exhaustion. On the other hand, when empyema is submitted to timely and adequate operation the prog-

[1] *Med. News*, 1890.

[2] *Deutsches Arch. f. klin. Med.*, vol. xlv.

[3] *Soc. méd. des Hôp.*, 1892.

nosis is excellent, providing, of course, there is no other incurable affection. Some recent statistics are surprisingly favorable: Morrison[1] operated 34 private cases in children with but 2 deaths; König[2] reports 76 successive cases of all sorts operated by himself in the course of ten years: of these, 10 died—*i. e.* 4 of pyæmia, 1 moribund on entrance, and the other 5 of causes quite independent of the pleurisy; of the remaining 66, 59 were completely cured, 3 were lost sight of, and 4 still had fistulæ. Runeberg[3] operated 61 cases from 1885 to 1890: 2 were tubercular and died with fistulæ; 10 were secondary to pulmonary gangrene, and of these 4 died; 1 died of erysipelas; of the remaining 48 uncomplicated cases, 46 were completely cured after an average duration of forty-eight days. Averaging the above three series would give a mortality of 10 per cent. in 171 unselected cases, and a complete cure in 93 per cent. of uncomplicated cases. This is undoubtedly optimistic, but it shows what may be accomplished by the modern methods of treatment. The individual case must be judged on its own merits. Up to the third year of life the prognosis of all forms is grave: Wightman[4] in a series of 124 cases of empyema in children lost 50 per cent. of those under three years of age. Unquestionably the most benignant form of empyema is that produced by the pneumococcus: nearly all cases in fairly healthy individuals recover when properly managed. Rupture into the lung does not materially affect the prognosis. Streptococcus empyema is, on the whole, a much less favorable variety, but here also, in uncomplicated cases, recovery may be confidently expected. Putrid forms are always grave, and death may occur within the first fortnight; not infrequently, however, the pus gradually loses its fetid character after operation and the case goes on to complete recovery. Tubercular empyema usually ends sooner or later in death, though this may be only after months or even years of fairly good health.

A word in reference to certain exceptional varieties and conditions. Spontaneous evacuation is likely to prove fatal if the pus has made its way below the diaphragm: above, through the chest wall, it somewhat impairs the prognosis, though not very materially. Runeberg's uncomplicated cases of spontaneous evacuation, among them 11 into the lung, all recovered after operation, and he is of the opinion that this event has but little effect upon the prognosis. Double empyema is exceedingly grave, but by no means desperate; numerous favorable cases have been reported, particularly in children, and it is at least a matter of great doubt whether, as formerly supposed, all double pleurisies are necessarily tubercular. In encapsulated forms the prognosis must depend largely upon the nature of the exudate and its accessibility; even when unoperated a favorable termination by vomica—as, for example, in the interlobar form—is not infrequent.

The outcome of hemorrhagic pleurisy depends more on the primary lesion than upon the mere presence of blood: the great majority of non-traumatic cases, being secondary to tuberculosis or cancer, are of very doubtful prognosis, though in the former recovery is possible. Pleural epilepsy and other cerebral complications of effusion are exceedingly grave, about half the reported cases having terminated fatally.

[1] *Lancet*, 1894.
[2] *Berliner klin. Woch.*, 1891.
[3] *Zeitsch. f. klin. Med.*, xxi.
[4] *Lancet*, 1894.

Treatment of Pleurisy.—*Dry Stage.*—It is usually impossible to determine at the onset of an acute pleurisy or for several days later whether or not effusion will develop; hence there can be no early distinction in treatment.

The first and, as a rule, most urgent indication is the relief of pain. For this purpose opium in some form is usually indispensable. Since a single dose is often insufficient, we prefer the hypodermic method, which permits of frequent repetition: it is, however, recommended by some to give gr. $\frac{1}{50}$ of morphine hourly by the mouth until pain is relieved. Local revulsive measures should accompany the narcotic. Sometimes it is best to begin by the application of three or four dry cups. Following these, a hot flaxseed poultice, to which mustard may be added, is a grateful application, though in some instances an ice bag gives greater relief. The poultice should be large, thick, and frequently renewed: Garland suggests for this purpose a bag of old flannel, which may be often refilled without acquiring a disagreeable odor. In place of these applications, or combined with them, it is often advisable to immobilize the side by strapping. Especially in the milder forms of dry pleurisy, such as so often accompany pulmonary tuberculosis, we have found this method of great service. Two or three 2-inch strips of rubber adhesive plaster may be used, applied during expiration so as to overlap the median line front and back; or, instead of plaster, a bandage may be applied, either a few turns of an ordinary elastic bandage or a cotton roller, as preferred by Otto, as much as possible of the well side being left uncovered. The results obtained by Otto seem to show that a well-fitting bandage is often a most valuable method of treatment in the acute stage of this disease.

A patient with acute pleurisy should be strictly confined to bed. This applies not only to the primary stage, but also to the subsequent period of development and increase of effusion. It is only when the latter has been for some time stationary, or, in large effusion, when absorption has already begun, that the patient should be allowed to go about. This point has been recently emphasized by high authority (Guttman; Senator; Volland); and the experiments of Dybkowski, showing that during the early stages there is an increased tendency to exudation when respiratory movements are active, point in the same direction. The bowels should, as far as possible, be kept somewhat freer than normal. It is hardly necessary to add that in some highly febrile cases we may at least increase the patient's comfort by cool sponging and by other means of temporary reduction of temperature.

Are there any forms of acute pleurisy which are amenable to a specific treatment? In those rare cases which are manifestly syphilitic rapid improvement may be expected from the use of mercury and the iodides.[1] Tubercular cases should, according to Velten,[2] be put upon large doses of creasote: we should prefer to at least restrict its use to subacute and chronic forms. Much greater interest attaches to the employment of the salicylates in cases associated with rheumatism, as well as in those which are apparently idiopathic. A glance at the etiology of pleurisy makes it evident that this medication must often prove futile, but the great benefit occasionally derived makes it appear justifiable to give

[1] Pretorius, *Annales de la Soc. de Méd. d' Anvers*, 1891. [2] *Berliner klin. Woch.*, 1893.

each case the benefit of any doubt. From a large number of favorable reports upon the use of salicylates in pleurisy we select the following: H. Köster[1] treated 27 cases of primary pleurisy with the salicylates, all cases of small or moderate effusion; the results of 17 of these are described as excellent—*i. e.* rapid fall of temperature, diminution of dyspnœa on the second day, increased diuresis even before any evidence of resorption, and rapid disappearance of the effusion. The usual dose of the salicylate of soda is ʒj to ʒiss per diem. I prefer to use it only during the acute stage, but its exhibition at a later period has authoritative sanction.

Sero-fibrinous Effusion.—During the acute formative stage of an effusion, while it is still constantly increasing, it is usually futile, in the absence of the *indicatio vitalis*, to employ any very active measures for its removal: one can only attempt to hinder the formation of an excessive quantity. This applies chiefly to typical cases where the stage of exudation lasts from ten to fourteen days. During this period the patient should be kept strictly in bed. His diet should be light and nutritious, the bowels opened two or three times daily by saline cathartics, and fever, if present, be partially controlled by sponging. In the majority of cases sodium salicylate is given, gr. x, every two to four hours throughout this period, intermitting, of course, with the appearance of deafness or tinnitus. On the flat area I begin the use of iodine or small fly blisters as soon as cessation of pain makes further poulticing unnecessary.

After the exudate has reached its *période d'état*—the height, namely, beyond which it shows little or no tendency to increase—it must be the prime object of treatment to assist nature in its removal. Such assistance may be rendered by—

I. **Medicinal Treatment.**—Locally the small fly blisters or the tincture of iodine should be continued. We have an especial preference for the following so-called Carson's paint:

℞. Olei tiglii, ʒss;
Ætheris, ʒj;
Tincturæ iodi compositæ, ʒiiss.—M.
Sig. Apply once or twice daily.

These paintings with iodine, made so as to include accurately the flat area, also serve to mark the original line of effusion.

Internal medication can be expected to accomplish appreciable results in only a limited number of small, or at most moderate, effusions. We believe that the best authority of the present day is strongly opposed to the prolonged use of such treatment exclusively, and to its continuance after a week or ten days have shown it to be inadequate. It is to be recommended only in comparatively robust individuals during the early stages of the période d'état, at the time when nature is endeavoring to effect a spontaneous cure. Such treatment may consist in either catharsis or diuresis. The former of these is to be considered by far the more efficacious method of the two, and the form to be preferred is the so-called method of Hays—*i. e.* catharsis with a dry diet.

[1] *Ther. Monatsheft.*, 1892.

The quantity of ingested liquids is limited to a pint or thereabouts in twenty-four hours, and saline cathartics are given, to the production of rather free watery stools. The rationale of the method is evident. The cathartic generally used is sulphate of magnesium, ℥ss or more, morning and night; Seidlitz powder is preferred by others. These cathartics should be administered in as concentrated a form as possible. This method is widely advocated for fairly robust individuals, and often effects rapid absorption.

Diuretics are less reliable—according to many absolutely useless. This is especially affirmed of the usual combination of infusion of digitalis with acetate of potash or squills. My experience with these remedies coincides with this view, and I have a preference for caffeine with the benzoate of soda, gr. ij of each in a capsule every two to three hours. Osler speaks favorably of diuretin (Knoll); it must be given in doses of ʒj–ʒiss per diem—a dosage, by the way, which sometimes makes it necessary to consider the question of expense. That an exclusive milk diet does, as is claimed, produce a diuresis which takes anything from the pleura is extremely improbable. It should further be mentioned that in suitable cases, chiefly subacute, the addition of a ferruginous or other tonic to the diuretic employed appears to intensify its effect. Fräntzel, for example, speaks of brilliant results in certain cases from the combination of decoctionis chinæ (4–6 per cent.) with acetate of potassium. Others recommend highly the syrup of the iodide of iron or the simple tincture.

II. **Aspiration.**—It seems unnecessary at the present day to undertake any defence of the operation of thoracocentesis in sero-fibrinous pleurisy. No one now believes that when ordinary care is observed it ever converts a serous into a purulent exudation, or that, except in the rarest instances, it need be responsible for sudden death. Thanks to the fruitful efforts of Trousseau, Dieulafoy, and Bowditch, every practising physician has now learned to perform this little operation with confidence and safety. The only question still under discussion are as to the exact conditions under which it is to be employed.

Indications.—1. When life is directly threatened or endangered—the so-called *indicatio vitalis.* Under these conditions immediate partial aspiration is demanded in all effusions of whatever size or character. The alarming symptoms may be those of pure asphyxia from compression, or of cardiac weakness as shown by a rapid and feeble pulse. Dyspnœa alone is not necessarily urgent, but if it is persistent and independent of pain, or especially if it tends toward even temporary attacks of orthopnœa, danger is surely at hand. Under any of these conditions delay is extremely hazardous, although it is both unnecessary and unsafe to remove more than a small quantity of fluid at once.

2. When the fluid has risen to the third interspace in front, no matter how rapidly it has formed or how little opportunity has been given for the trial of medicinal measures. On this point there is nearly as great unanimity among authorities as upon the *indicatio vitalis.* By early operative interference not only is a dangerous increase forestalled, but a step is taken which experience has shown would have been ultimately necessary, since effusions of this size rarely disappear spontaneously within a reasonable period.

3. In all lesser effusions when spontaneous absorption is unduly delayed. Just how long we should wait is a question to which different answers have been given: all, however, agree that there is less risk in haste than in delay. No inflexible rule can be laid down which is applicable to all cases. We have seen that a période d'état of several days' duration is, so to speak, a normal incident of acute pleurisy. When, therefore, an effusion has pursued a typical course of acute rise (one to two, rarely three, weeks), followed by a stationary period, I would wait until the latter has lasted at least seven days before aspiration. In atypical cases, where the amount of fluid oscillates or tends to steadily rise, it is best to puncture in about three weeks from the date of onset. In a case with indefinite history, seen for the first time with established effusion, I would aspirate at once, or in a tolerably robust individual wait perhaps a week at most for the trial of internal remedies. In general, the greater the debility, dyspnœa, or cardiac weakness, the earlier should aspiration be performed. There is no absolute contraindication to the operation.

Technique.—The apparatus in most general use for paracentesis, and on the whole to be preferred, is the familiar bottle aspirator of Potain. A large bottle is connected by a couple of rubber tubes, each provided with a stopcock, both with an aspirating needle and an air-pump; by the latter the air in the bottle is partially exhausted, so that suction is exerted upon any fluid into which the needle may be plunged. This apparatus is simple, easily kept in order, and its action is steady. Robinson[1] recommends the Allen pump. Bowditch preferred the simple Dieulafoy barrel, chiefly because of the accuracy with which the operator can estimate the amount of suction employed. The needle used in aspiration should be very small: it need not necessarily be more than ½ mm. in diameter, though I prefer one of about double this size—equal to that of a No. 4 French bougie. This matter of the size of the needle is extremely important, not so much as regards the avoidance of pain as because of the necessity that the fluid should be removed very slowly and gradually. In exceptional cases of thick, grumous exudate the small needle may have to be withdrawn and a larger one substituted, though it is surprising how rarely this is necessary. A sharp hollow needle is generally preferable to a trocar, both because of its smaller calibre and because its introduction is less painful.

The site of puncture is not a matter of very great importance. It must be high enough to avoid the costo-diaphragmatic gutter—two to three inches at least above the lower boundary of the lung, as best determined by percussion of the opposite side—and for manifest reasons a considerable distance below the upper level of fluid. In small effusions it may be about halfway between the spine and the posterior axillary line; in a larger effusion the axillary region is generally preferred at the fifth or sixth space or at a point farther forward and a little higher, though still outside the mammary line. The patient may be operated on in the sitting posture or semi-recumbent upon the edge of the bed and slightly rotated toward the affected side (Powell); Bowditch usually preferred that he should sit sideways in a chair with arm resting upon the back.

[1] *Med. Record*, 1893.

The principles of asepsis must be strictly observed in aspiration. The patient's skin at the site of puncture and the hands of the operator must be thoroughly sterilized with brush, bichloride, and ether as for any more extensive surgical procedure. The needle is boiled for twenty minutes, and the proximate tube of the aspirator soaked for one hour in a 5 per cent. solution of carbolic acid. If care is taken in these particulars, aspiration need never be responsible for the conversion of a serous exudation into pus.

General anæsthesia is never employed for simple aspiration: it is not only unnecessary, but objectionable. Locally, an injection of cocaine, or an ether spray, or even the application of ice and salt,[1] may profitably be used to lessen pain. It is also a customary and commendable practice to give an ounce of whiskey before operating.

Having carefully located the intercostal space with the tip of the left index finger, the needle, once entered, is thrust vigorously forward into the pleural cavity. If the hollow needle is used, a preliminary incision through the skin is unnecessary. Any boring motion of the instrument is to be avoided. The advice is usually given to keep close to the upper margin of the lower rib, in order to avoid the intercostal artery: I consider this danger somewhat chimerical, and prefer rather to penetrate as nearly as possible in the middle of the space, since pain is much increased by any scraping of the bone. Often the space is so narrow that it is necessary to advance the needle with considerable caution until it has once engaged between the ribs, when the thrust may be more vigorous. A sense of diminished resistance usually tells the operator that the needle has entered fluid.

How much fluid shall be withdrawn? The first consideration must always be the immediate effect of aspiration upon the patient. Bowditch used to say, "Tell me as soon as you feel the slightest discomfort—either pain or sense of constriction or desire to cough;" in either event he either temporarily suspended the aspiration or, in case of recurrence of the symptoms, withdrew the needle. I can suggest no better rule as to the minimum quantity to be removed. As to the maximum, while it is true that immense quantities—126℥ from one pleural cavity—have been removed with impunity at a single sitting, it is also certain that large aspirations have been responsible for a considerable proportion of the reported cases of sudden death and pulmonary œdema. For this reason especially, and also because a small aspiration is usually sufficient to inaugurate the rapid absorption of an effusion hitherto stagnant, moderation is imperative. I consider the following a good general rule: In most cases be content with the removal of two pints; never withdraw more than three pints at a single sitting, even where no ill effects are apparent and it seems absolutely safe to proceed. Netter advises the removal of 1000 c. c. (about two pints) every alternate day until the chest is emptied; and it is certainly far better to repeat the aspiration several times, if necessary, than to subject the patient to any risk. If the above quantity be not exceeded, there can be no objection to wholly emptying the chest at once, provided there are no subjective symptoms of distress. But complete removal is no special desideratum,

[1] Vide Treatment of Empyema, p. 305.

and never to be sought at the expense of the least danger, or even discomfort, to the patient.

After the needle is withdrawn it is unnecessary to apply any dressing to the wound other than collodion or a bit of adhesive plaster. For at least twenty-four hours after the aspiration the patient should be strictly confined to bed. Powell warmly favors a firm strapping of the side immediately following the operation, believing that it will hinder a re-accumulation; in view, however, of Dybkowski's experiments as to the favorable effect of respiration upon pleural absorption, it may well be questioned whether such strapping might not have the opposite effect and seriously delay the final and complete removal of the residue of fluid.

The danger of aspiration as above described, and as very generally practised at the present day, is exceedingly small. At the same time, it cannot be too strongly insisted upon that careless aspiration is fraught with risk. The operation has now become so universal that the tendency of late years is perhaps to regard it too lightly. We cannot afford to disregard the bitter experience of our predecessors with large cannulas and abundant aspirations. Deaths from pulmonary œdema—both of the compressed and of the opposite lung—from pulmonary embolism, cardiac thrombosis, and syncope were not so very infrequent when it was common to rapidly withdraw large quantities of fluid. The sudden filling of the paralyzed vessels of the compressed lung, and the almost violent return of the heart and great vessels to their normal position, which may result from rapidly emptying the chest, easily account for these frequently fatal sequelæ of thoracocentesis. The slow removal of only a moderate quantity seems to be that feature of the present method which ensures its almost absolute safety. Dieulafoy could find no case of death from aspiration in which not more than 1200 c. c. were withdrawn. If, therefore, 1000 c. c. are not exceeded; if this amount is removed slowly and cautiously; if any appearance of distress, such as constriction, pain, dyspnœa, violent cough, or syncope, is regarded as an imperative signal of danger,—aspiration may be performed, as shown by the statistics of Bowditch, Mason, Fräntzel, and others, in an indefinite number of cases without a death.

Other risks attendant upon the operation are extremely slight. Instances of accidental puncture of the liver or spleen were without harmful consequences. The danger also of seriously wounding the lung is very small: cases are numerous where it has been inadvertently pricked with perfect immunity. The entrance of air into the pleura is hardly possible with careful management of the aspirators now used; it is of course to be carefully avoided, but even should it occur it is very doubtful whether this alone would ever result in the development of pus; it certainly does not, as a rule. With sterile instruments and proper care aspiration is a harmless procedure and a most valuable means of relief and cure.

Hitherto I have spoken only of growing or stationary effusions, but there are also other possibilities to be considered. An effusion may be perceptibly on the decline, and yet the process be extremely slow and tedious. Such cases often need tonics, and the addition of a good preparation of iron to the diuretic or other treatment employed is often fol-

lowed by the happiest results. In other cases absorption may be furthered by increased pulmonary exercise, especially in combination with a change of climate. I have already alluded several times to the conclusions reached experimentally by Dybkowski, that the absorption of an effusion takes place almost wholly through the costal pleura, and that the respiratory act is largely concerned in the process. Nothing, therefore, could so meet the indications in many cases of slowly diminishing effusion as the moderate stimulus to respiration and the general tonic influence incident to residence in a mountainous and elevated region—such, for example, as the Adirondacks, Davos, and particularly Colorado and Arizona. The same is also true of cases more distinctly convalescent, where all fluid may be presumed to have been absorbed; few of these would fail to be benefited by the removal for a few weeks or months to a higher altitude. When circumstances render this impossible substitutes must be sought for: we must endeavor to assist nature in effecting a perfect re-expansion of the lung. To this end mild gymnastics are indicated, as, for example, with light Indian clubs or pulley-weights, and especially walking or even running. A method of systematic exercise in expiratory expansion, first suggested by W. B. James and highly recommended by Osler especially for operated cases of empyema, is the following: Two one-gallon bottles are filled with tubes like an ordinary Wolff bottle, and connected so that by blowing into one the water contained in it is forced into the other; from this it may then be driven back in like manner into the first bottle. Only one bottle is emptied daily at the outset, but a steady increase can be made according to the patient's strength.

But even when the last remnant of an effusion or its effects has entirely disappeared the task of the physician is not yet accomplished. He should regard his patient as peculiarly liable to the development of pulmonary tuberculosis. In many cases of poor physique, doubtful antecedents, or unfavorable surroundings the only unquestionable course to pursue would be to advise a permanent change of residence to a more favored climate. At least the patient should be kept under close observation: any depreciation of the general health, and particularly any suspicion of an apical catarrh, should be the signal for instant departure, or, if circumstances forbid, for a hygienic régime the chief factors of which are superalimentation and an out-of-door life.

Treatment of Empyema.—Whenever in any case it becomes evident that the pleura contains pus, its immediate removal by operative procedure is invariably indicated. Practically, only the following three methods need be considered:

1. *Aspiration.*—For the great majority of empyemas aspiration is entirely inadequate as a means of cure. There can be no doubt, in view of its abuse in this affection, that the mortality of empyema would be greatly lessened if it were always laid down as an inflexible rule to incise and drain. Nevertheless, such a rule would unquestionably do violence to the truth for the benefit of the careless and undiscriminating physician. Certain cases do recover after simple aspiration, the residue of pus undergoing complete absorption with apparent *restitutio ad integrum* of the affected side. I believe, therefore, that a *single* aspiration is justifiable under the following conditions:

(1) In response to the *indicatio vitalis*. When immediate danger threatens, as from compression or œdema of the lung, and when, because of great weakness of the patient or for other reasons, the radical operation cannot be at once performed. Here aspiration is merely a temporary expedient, to be shortly followed by a permanent opening.

(2) In the empyema of infancy or early childhood, whenever, owing to a fair general condition, no risk is incurred in a slight delay. A large proportion of these cases, though not manifestly metapneumonic, are pneumococcal, and show, therefore, but little tendency to become septic. It is true that most of them will eventually come to further operation, but if even a small proportion can be cured by so simple a procedure as aspiration, it would seem wise in proper cases to make the trial. It is to be strongly emphasized that such trial is permissible only in the absence of all threatening symptoms, and that in case of failure but a very few days should be allowed to elapse before resorting to the radical operation. As evidences of failure we should regard a rapid re-formation of pus or persistence or immediate return of a high degree of temperature. If, on the other hand, as I have personally observed in the case of a child of four, aspiration is at once followed by a pronounced and permanent fall of temperature, with return of appetite and a constantly increasing general euphoria, any further interference may be safely deferred. The younger the child, down perhaps to the second year, the better are the prospects of success.

(3) In those adult empyemas which are manifestly pneumococcal it is, perhaps, to be considered justifiable to pursue the same course as in those of infancy and childhood. This is the teaching of the latest French writers (Debove, Courtois-Suffit, Netter; also L. Ferdinand), and a very considerable number of favorable results have been reported. Netter, for example, has seen 10 cases of empyema in adults cured by simple aspiration. It is claimed for the method not merely that it is milder, but especially that it ensures an earlier and more complete expansion of the lung.

I would consent to even a single aspiration only when the amount of pus is small, the general condition excellent, and the etiological evidence conclusive. Netter insists that there should be no mixed infection, but a pure culture of the pneumococcus. Under such conditions the procedure is possibly admissible, though even here general opinion is at the present time almost unanimous in favor of the radical operation.

In those chronic and latent forms of empyemas which are manifestly tubercular it is often an open question as to the best course to pursue. The results of free incision in such cases are extremely discouraging. Not only are recoveries very rare, but there can be no doubt that life is frequently shortened by the exhausting and often subsequently putrid discharge. Moutard-Martin lost all of 7 operated cases. Krönlein had but 4 recoveries in 15; and so the list of unfavorable results might be multiplied—whereby it is also to be stated that in many reported series there was no bacteriological proof of tubercular empyema, but its existence was simply inferred from the fact that the affected individuals were phthisical. On the other hand, when not submitted to operation these tubercular cases often run an extremely slow and relatively benignant course. It is therefore not surprising that there are high authorities

(Netter, Guttman, Senator, and others) who advocate repeated aspiration as the most serviceable and conservative procedure. It will be observed that reference is here made to genuinely tubercular cases of chronic character, and not to those which are merely complications of pulmonary tuberculosis: many of the latter are non-tubercular, and not infrequently of more or less acute onset and course; such cases—usually streptococcal—should of course be at once submitted to the radical operation. In the genuinely tubercular form one must sharply individualize. If the lungs are known to be seriously diseased and there is little or no hope of recovery from the pulmonary affection, little can be said in favor of anything more than palliative aspirations. If the lungs are sound or nearly so, much depends upon the ability of the compressed lung to re-expand, and the correspondingly diminished chances of a permanent fistula. Bäumler[1] advises in such cases a preliminary aspiration. If on withdrawal of 1000 c. c. or more of pus there is no pain or marked tendency to cough, no cyanosis or smallness and frequency of the pulse, he would infer that the lung is still capable of expansion, and would soon venture the radical operation. If, on the contrary, by the development of the above symptoms the lung should prove itself incapable of expansion, he would repeat the aspiration at longer or shorter intervals, and be guided as to further measures by the degree of reaction to each aspiration and of improvement in general condition. In case of satisfactory progress he would finally attempt a permanent cure, either by the Bülau method as being mildest, or possibly even by thoracotomy. In intermediate forms, where there is evident phthisis, but no great improvement of general health, it is more difficult to decide upon the best course to pursue, and it must be left with the individual operator to follow the counsels of his own experience.

In all cases not included under the foregoing conditions, and even in these, except the first, if the operator be so disposed, immediate free evacuation and drainage are indicated, whatever the amount of pus or the general condition of the patient. There is no contraindication. Even where there is an external opening the usual operation should be performed and the fistula itself opened up and scraped. There is a difference of opinion as to the best site for incision. By some the eighth or ninth interspace behind, just below the angle of the scapula, is preferred, as affording the most perfect drainage in the usual recumbent posture of the patient. But this location is open to the objection that the ribs are here closer together, the chest wall thicker, and especially that a subsequent rise of the diaphragm is apt to bring the end of the drain into the costo-diaphragmatic gutter. Most operators therefore choose the mid-axillary line from the fourth to the sixth rib, and experience seems to show that an opening at this site ensures perfectly satisfactory drainage.

As to the operation itself, opinion in this country seems pretty definitely fixed. It is extremely rare that any other method is followed than that of either simple incision or incision with resection of a small portion of a rib. The so-called Bülau method—that of siphon drainage—has found but little favor outside of Germany, although it has given some excellent results and is warmly advocated by such men as Cursch-

[1] *Deutsches med. Woch.*, 1894.

mann, Immerman, and Leyden.[1] It consists, in a word, in puncture with a large trocar and cannula, through which a Nélaton catheter is introduced into the chest; the cannula is then withdrawn, leaving the catheter to remain permanently in situ; to the latter is attached a long rubber tube ending in a small bottle partly filled with an antiseptic fluid: this bottle may then be carried about by the patient, and is sometimes provided with a second tube through which suction may be made in case of obstruction. It is claimed that the siphon ensures a constant and satisfactory drainage, and that the absolute exclusion of air greatly favors re-expansion of the lung. On the other hand, the method is objected to on the ground that the catheter often gets occluded; that it is apt to become loosened, so that air enters about it and destroys the siphonage; and that, after all, the drainage through so small a tube must often prove insufficient, particularly when the exudate is full of clumps. Again, on account of the constant care demanded, the method is manifestly inapplicable to children. It is, however, to be stated that good results were obtained by Curschmann in 63 out of 75 cases thus treated—by Immerman in 57 cases, 49 complete cures.[2] It is quite possible that under more precise indications than can at present be established the method will in the future be widely recognized as the best for certain cases.

2. *Thoracotomy.*—The only advantage of simple incision over resection is that it is, on the whole, a more trivial operation, and can be done, if necessary, with only local anæsthesia. It is therefore to be preferred in certain cases in adults where general anæsthesia would be dangerous; and by very common consent it is the most desirable operation for most cases in children, although here, when there is no contraindication, most operators use a general anæsthetic, preferably chloroform because it ensures a quieter respiration than ether, and therefore less danger of a possible rupture of the empyema into the lung during operation. For local anæsthesia an ether or rhigolene spray may be used, or the subcutaneous injection of cocaine; or a piece of ice, a couple of inches square and with flat surface, may be dipped in salt and applied to the chest for twenty to thirty seconds (Powell). Operation should always be preceded by an exploratory puncture. The incision is made, under the usual antiseptic precautions, in the middle of the selected interspace, about two inches in length and down to the intercostal muscle; the pleura may then be reached, either by gradual dissection or by the use of a grooved trocar along which the knife is thrust rapidly into the cavity. This incision through the muscles and pleura need not be over an inch in length, and in order to avoid the intercostal artery should be as close as possible to the lower rib. After most of the pus has been allowed to slowly escape a good-sized drainage tube, guarded externally by a large safety pin, is inserted so as to project not more than two or three inches into the cavity. The whole side, including the arm, is then covered with a regular surgical dressing, particular care being taken to thickly pad the axilla.

3. *Thoracotomy with Resection.*—For all cases in adults where a general anæsthetic can be administered the resection of one or two inches of a single rib has very decided advantages over simple incision. These

[1] *Verhandl. d. Internat. Cong.*, 1890. [2] Loc. cit.

advantages appear to consist almost wholly in the freer drainage afforded by the larger opening, facilitating especially the ready escape of the fibrinous clumps so frequently present. Beck [1] considers it also extremely desirable to be able to insert the finger and sweep it about for the purpose of freely opening up at least the immediate neighborhood of the drain. Laache,[2] again, in the choice of resection puts especial weight upon his observation that the more perfect drainage ensured by this method obviates the necessity of subsequent irrigation with its attendant dangers. Whatever may be the explanation, it must be admitted that resection has given, in general, better results than simple pleurotomy, and that at the present time it is preferred by the great majority of operators in all lands. Simonett [3] found that in 79 cases of resection collected by him there were 55 complete cures, while in 107 cases of incision alone only 60 recovered. The remarkable results also obtained by Runeberg and König (vide p. 321) were all in cases of resection. The subperiosteal removal of one or two inches of a single rib is an operation unattended with danger, producing no deformity, and in most cases probably followed eventually by a complete restitution of the excised bone. General anæsthesia is usually necessary, and by many operators chloroform is preferred: the danger of an anæsthetic in most adult cases of empyema does not appear to be much greater than in other conditions.

The operation may, in brief, be performed as follows: An incision some three inches in length is made down to the middle of the sixth or seventh rib in the axilla. The periosteum is then crowded to either side by the aid of an elevator, the rib firmly grasped with a strong forceps, and an inch and a half excised by one of the various rib-cutters or an ordinary Liston bone forceps: it is a considerable advantage to have avoided penetrating the pleura while disengaging the rib. After the latter has been removed the pleura is incised, and one or two fingers are inserted in order to regulate the flow of pus, which should not be too rapid: the finger is also swept about within on all sides of the incision for the purpose of thoroughly opening up its immediate neighborhood. The wound is then drained and dressed as in simple thoracotomy.

The operation of the special forms of empyema differs but little from that of a typical case. It has already been said that in pulsating empyema or external fistula it is generally best to make a fresh incision in the usual situation. The same is true when rupture has occurred through the lung: no time should be lost in waiting for spontaneous recovery. In double empyema it is usually preferred to allow several days to intervene between the two operations.

The after-treatment of empyema is so frequently in the hands of the general practitioner that it seems desirable to refer more in detail to a few important particulars.

Irrigation has long been regarded as a most salutary and essential feature of the management of an empyema. Of late, however, the opinion has been steadily gaining ground that except in putrid cases all irrigation, even at the time of operation, should be omitted, as being not merely unnecessary, but harmful. Aside from the occasional grave accidents which attend its use (vide Complications, page 290), it seems that

[1] *Med. Record*, 1893. [2] *Deutsches med. Woch.*, 1894.
[3] *Thèse de Bâle*, 1889.

it tends to actually retard recovery. Runeberg[1] at the clinic of Helsingfors made the following remarkable series of observations, already briefly referred to: From 1876 to 1883, when constant lavage was employed during convalescence, there were 30 per cent. of cures and an average duration of 101 days; from 1883 to 1885, under a single washing at the time of operation, there were 70 per cent. of cures, average 84 days; from 1885 to 1890, with absolutely no irrigation, the cures, in uncomplicated cases, reached 96.5 per cent., and the average duration was only 48 days. While admitting the influence of other factors, Runeberg ascribes this improvement very largely to the suppression of irrigation. This accords also with the present practice of other eminent surgeons: Cabot and Lindsay irrigate only once, at the time of operation; among those who have entirely done away with lavage in ordinary empyema are Bucquoy, Morrison, Holt, Laache, Beck, and König. The results obtained by the latter operator[2] are so remarkable that it may be well to give his method in detail. His object is to effect the most perfect drainage possible. To this end the patient is instructed to lie as much as possible on the affected side. He is also subjected to a series of manipulations: at first four times, and later but two or three times daily, he is lifted by the legs and hips so as to rest for a moment on the shoulder of the affected side; he then assumes for a short time the semi-recumbent, partly rotated posture; and, finally, he is lifted again as before. This same drainage from below may also be effected by the patient alone, who allows his shoulders to hang over the edge of the bed, supporting them with the hands upon the floor. Certainly nothing can be said against the simplicity and reasonableness of this method, and König's results seem to speak strongly in its favor.

In putrid empyema irrigation is still employed by most surgeons, although even here it would appear that an entirely free drainage without lavage is often sufficient to rapidly alter the character of the pus (Hertz). Most operators prefer to wash thoroughly at the time of operation with a very mild antiseptic—usually a saturated solution of salicylic acid or a 3 per cent. boric-acid solution—and to repeat this daily until all fœtor has disappeared.

Another consideration of importance is the management of dressings. König uses a very large and bulky occlusive dressing, thickly padded in the axilla and including even the arm. He generally finds it necessary to change the first dressing on the second or third day, but the subsequent dressings are frequently left for a week; the guide, in general, is the thermometer and the appearance externally of pus. Cabot recommends that a sheet of mackintosh be included in the dressing, believing that he thus favors a valve-like action of the latter which readily permits the escape of air, but prevents its entrance, so as to directly promote the re-expansion of the lung: it seems probable that any modern dressing exerts at least a certain tendency in this direction. The drainage tube should be taken out every few days and thoroughly cleansed; in its shortening and final removal the same rules are to be observed as would govern the management of any other suppurating cavity.

If there are no complications and but little or no elevation of tem-

[1] *Zeitschrift f. klin. Med.*, xxi. [2] Loc. cit.

perature, the patient may be allowed to go about after the first week. Every effort should be made to improve the general nutrition. The lung should be helped to expand by the use of the Wolff bottles, as described in the treatment of simple effusion (page 316), and by such exercise as is adapted to the patient's strength. It is hardly necessary to suggest the frequent desirability of tonics and the benefit often to be derived from a temporary change of climate during convalescence. When operation has been too long deferred, or when from other causes the lung shows but little tendency to expand and it becomes evident that the pus cavity can only become obliterated by marked retraction of the chest wall, we may attempt to aid this process by the long-continued use of a firm strapping over the affected side. For Estlander's and other more serious operations, which must be taken into consideration when simple drainage has failed to cure, we must refer to works on surgery. In chronic fistulæ, associated with advanced pulmonary or general tuberculosis, little can usually be done other than to try and prevent secondary putrid infection of the suppurating cavity by scrupulous care in the management of dressings. In these old and hopeless cases of chronic fistula we have found an oakum pad over a small wad of iodoform gauze a dressing which is both inexpensive and satisfactory.

HYDRO-THORAX.

Definition.—This term is applied to a simple dropsical transudation into the pleural cavity, as distinguished from an inflammatory effusion.

Pathology.—A simple hydro-thorax is always a secondary affection, and usually one of the numerous manifestations of general anasarca. It may, therefore, with the latter, follow chronic obstructive conditions of the lesser circulation, oftenest of cardiac, sometimes, as in chronic emphysema, of pulmonary origin; also any form of hydræmia, whether produced by renal disease or by some one of the various cachexias. In very rare instances a hydro-thorax may be independent of general dropsy, and the result of intra-thoracic growths which by pressure hinder the return of blood or lymph from one or both pleuræ. Hydro-thorax is almost always double, though the fluid usually predominates on the side upon which the patient habitually lies. It will be unilateral if the opposite pleura is obliterated by adhesions; and Osler states that it is often so in cardiac affections, though this does not seem to accord with general experience. It is almost invariably a late development of anasarca, and it seems extremely doubtful whether, as claimed by Fernet and Niemeyer-Seitz, it is ever the earliest manifestation of dropsy. The pleural membrane is found unaltered, except that it is somewhat œdematous and its transparency is slightly diminished. The lung is retracted or compressed as in ordinary effusion, and other organs are also correspondingly displaced. The fluid itself is usually found in but moderate quantity, though it may be very abundant; it is of pale yellow color, contains much less fibrin and albumin than an inflammatory exudate, and no flocculi or leucocytes; it never coagulates spontaneously.

Symptoms.—The symptoms of hydro-thorax, as such, are, subjec-

tively, hardly more than a varying degree of dyspnœa : this, however, is very apt to be excessive, both because the transudation is bilateral and also because other sources of dyspnœa are rarely wanting in these cases. There is no pain or fever, and other local or general symptoms are due to the primary disease. The physical signs are in the main those of pleuritic effusion. It is said that the fluid is considerably more mobile in hydro-thorax, and responds more quickly to the influence of gravity. Cardiac displacement is of course lacking in double hydro-thorax, and that of the diaphragm too, often hindered at least by the accompanying ascites.

The DIAGNOSIS must depend almost entirely upon the double character of the transudate, and especially upon its association with general dropsy.

The PROGNOSIS and TREATMENT also depend almost wholly upon the primary affection. When general dropsy is extreme Fräntzel[1] has found the insertion of several hypodermic needles into the subcutaneous tissue of the legs to be the most effectual method of reducing the hydro-thorax ; the needles are left in situ, and to them rubber tubes are attached through which the fluid slowly drains off in large quantities. The failure of such methods and of medicinal treatment, and especially the development of great dyspnœa, demands the withdrawal of a portion of the fluid by aspiration, which must then, of course, be repeated at frequent intervals. (For the technique of aspiration we refer to the section on Sero-fibrinous Pleurisy, page 299.)

PNEUMO-THORAX.

DEFINITION.—A condition characterized by the presence of air in the pleural cavity. The terms pneumo-hydro- and pneumo-pyo-thorax are used to indicate the presence also of serum or pus.

ETIOLOGY.—The old idea that gas is sometimes secreted by the pleura may here be summarily dismissed. That gas may exceptionally develop in the pleural cavity from decomposition is still held by good authorities (Senator, Biermer, Weil); and a recent observation of Levy,[2] demonstrating the presence of an anaërobic gas-forming microbe in a pneumo-thorax which followed pleuritic effusion, lends confirmation to this belief. Practically, however, in any given case of pneumo-thorax it may safely be assumed that the pleural cavity contains air which has gained entrance from without by perforation.

Among the various causes of perforation the first naturally suggested is trauma. Pneumo-thorax may result, not only from any penetrating wound of the costal or visceral pleura, but also from severe contusions of the thorax, such as attend a fall from a considerable height, a heavy blow, compression between two cars or beneath a heavy wagon-load, and the like : here air enters the pleural cavity through a perforation of the visceral pleura, caused either by the sharp end of a broken rib or by simple bursting of the lung in consequence of sudden and violent

[1] *V. Ziemssen's Handbuch*, "Hydro-thorax."
[2] *Archiv. f. experimentelle Path. und Pharmakol.*, 1895.

compression. Penetrating wounds of the thoracic wall do not, however, necessarily result in pneumo-thorax. West [1] has shown that a very considerable degree of cohesion exists between the opposed pleural surfaces, stronger even under certain conditions than the retractile power of the lung. Hence more than a mere prick of the pleura appears to be essential for the production of immediate collapse of the lung, although H. Biggs has reported 2 cases where pneumo-thorax was caused by simple puncture with a hypodermic needle: such an occurrence is certainly very exceptional in proportion to the great number of instances where the lung has been accidentally wounded in exploratory puncture.

Aside from trauma, the primary cause of pneumo-thorax in an overwhelming majority of cases is pulmonary tuberculosis. If empyema be excepted—responsible for perhaps 5 per cent. of all cases—it may be affirmed that other forms of pneumo-thorax than the tubercular are among the greatest exceptions. According to Weil,[2] at least 90 per cent. of all cases are tubercular. As a rule, the perforation appears to be due to the softening and rupture of a fresh tubercular nodule lying in or immediately beneath the pulmonary pleura, and not, as was formerly supposed, to the rupture of a large cavity. Weil was among the first to call attention to the frequency of pneumo-thorax in the earlier and more recent forms of phthisis; and, although it does often occur late in the disease, even then it is rarely associated with an old cavity. The adhesive pleurisy which attends the development of the more chronic destructive processes in the lung is unquestionably conservative, and accounts for the comparative infrequency of pneumo-thorax in pulmonary tuberculosis. Hence also the usual site of the perforation in the lower part of the upper lobe, rather than higher up where old adhesions have shut off all possible communication between large pulmonary cavities and the cavity of the pleura. Tubercular pneumo-thorax is found twice as often on the left side as on the right, and, like the disease from which it springs, is most common between the twentieth and fortieth years of life. It occurs very much oftener in men than in women. Its frequency in pulmonary tuberculosis is estimated by most authors at about 5 per cent. Weil, however, found that in 355 autopsied cases of phthisis no less than 10 per cent. were complicated by pneumo-thorax—certainly a more accurate method of estimation than the mere clinical figures usually given.

Empyema is universally ranked next to phthisis in etiological importance, causing probably about 5 per cent. of all cases of pneumo-thorax. In the great majority of such cases air enters the pleura from the bronchi through a visceral perforation; an external opening is so often sinuous and of a valvular nature that pneumo-thorax rarely follows spontaneous evacuation through an intercostal space. Nor is it, indeed, to be supposed that a pneumo-thorax always and necessarily results from vomica: here also it is probably the exception rather than the rule.

An uncommon but exceedingly interesting and important form of pneumo-thorax has been termed "accidental:" it is characterized by occurrence in apparently healthy individuals and by a usually favorable

[1] *Brit. Med. Journ.*, 1887. [2] *Zur Lehre vom Pneumo-thorax.*

course. While cases apparently accidental are often due to a latent tuberculosis, it is now definitely conceded that pneumo-thorax is occasionally caused by the simple rupture of a healthy or, more frequently, emphysematous lung. Gaillard[1] has collected 37 cases of this variety, and in the 3 only of these which came to autopsy was there found in each a ruptured emphysematous bulla. The form occurs oftenest in connection with the vicarious or localized emphysema of young adults, much less frequently in the chronic emphysematous—the inveterate form—or with the pertussis of children. This apparent paradox is attributed by Gaillard to differences in the subpleural connective tissue at different ages, its firmer character in the young adult favoring the simultaneous rupture of both pleura and the adjacent pulmonary tissue. The immediate cause of most cases of accidental pneumo-thorax—as, indeed, of all other forms—is some violent respiratory effort, such as a fit of coughing, heavy lifting, or severe straining at stool; but it is worthy of special note that not infrequently the onset has been preceded by no such exertion, and has even occurred during sleep.

Other causes of pneumo-thorax are so infrequent as to hardly require more than simple enumeration. They are all destructive processes, and may be either visceral or parietal. Among the former pulmonary gangrene is relatively common and produces an especially fatal form. Among 918 cases of pneumo-thorax collected by Biach, gangrene was noted as the cause in 65—a figure which is, however, undoubtedly far above the usual proportion. Pneumo-thorax may be caused by the rupture of a pulmonary hydatid or of an abscess due either to pneumonia or an embolic infarction. It may be secondary to cancerous processes of the lung or œsophagus. In several instances it has been produced by the attempt to force a bougie through an œsophageal stricture. A bronchiectatic cavity may break into the pleura, the resulting pneumo-thorax being always putrid and generally fatal. Parietal sources of pneumo-thorax are peripleuritic and glandular phlegmons and cancer of the breast or axillary glands. Certain abdominal affections may lead to pneumo-thorax, oftenest ulcerative processes of the stomach or colon; also abscesses, as of the liver, which may first find their way into the lung and subsequently into the pleura.

Pneumo-thorax in children is rare. It oftenest originates in a broncho-pneumonia from the rupture of small superficial abscesses, such as the French have termed vacuoles; Rilliet and Barthez assert that pneumo-thorax from this cause is not so very infrequent in children under seven years of age. Cnopf has also recently called attention to its occurrence as a complication of diphtheritic croup, and reports 4 cases of this sort; he attributes it to the rupture of either an acute emphysematous bulla or an hemorrhagic infarction. Finally, a suppurating bronchial gland may open first into the pleura, and subsequently into the trachea or œsophagus. Very rarely pneumo-thorax in children may be tubercular or originate in other of the conditions which have been referred to.

Pathological Anatomy.—Pneumo-thorax usually involves the whole pleural cavity of one side, very rarely of both; exceptionally it is limited by old adhesions, and is then spoken of as partial. It may

[1] *Du Pneumo-thorax simple sans Liquide, etc.*, reprint, 1888.

also vary greatly according to the character of the perforation. If the latter permits the free entrance and exit of air, the pneumo-thorax is said to be open, and intra-pleural pressure is of course simply atmospheric. In closed pneumo-thorax, on the other hand, the perforation has become occluded, and here pressure may be either positive or negative according to the original character of the perforation and the progress of absorption. Finally the perforation may be valvular, permitting the entrance of air with inspiration, but closed in expiration. In this form the confined air may be under very high tension. This excess of air is due at first to the pumping effect of inspiration: later a still further excess may probably, as claimed by Bouveret, be forced into the already distended pleura by the violent expiratory efforts of cough.

At the outset most cases of pneumo-thorax are undoubtedly valvular. Powell[1] examined 16 cases with reference to intra-pleural pressure, and found that in 12 of these it was from one to seven inches of water above the atmospheric, showing that in at least 75 per cent. the perforation had originally been valvular.

In all forms of pneumo-thorax except the partial the affected side is found very considerably distended, with more or less obliteration of the intercostal spaces. On puncture of the pleura the air, if under positive pressure, will escape with more or less perceptible force. The composition of this air is similar to that of the external atmosphere. The longer it remains in the closed pleura the greater is the proportion of carbonic acid and the less that of oxygen—a fact which Ewald would utilize in the diagnosis between the open and closed forms. When the pleural cavity is opened the lung, if free, is found compressed against the spine to a small bluish or brownish fleshy mass not much larger than the closed fist. It contains no air after the lapse of a few hours from the time of perforation, though it can usually, in recent cases, be readily inflated through the trachea to its original size. There is often enormous displacement of mediastinum and diaphragm. In left pneumo-thorax the whole heart may be found to the right of the median line; and when the right pleura is involved the liver may be wholly below the free borders of the ribs. It is especially to be noted that even in open pneumo-thorax the degree of displacement is still very great, as has not only been demonstrated by the experiments of Powell[2] and Weil, but as one would be led theoretically to expect. The mediastinum, for example, is normally under the influence of the negative pressure of both pleural cavities: substitute for this on one side atmospheric pressure, and the mediastinum must of course be crowded over toward the well side until its increasing tension neutralizes the excess of unilateral pressure. The same is true of the diaphragm: below it is intra-abdominal pressure; above, the negative pressure of the pleura; increase the latter while the former remains unchanged, and the diaphragm must necessarily descend. The point often so strongly insisted upon (Powell, Garland, Donaldson) that the mediastinum is drawn over by the retracting lung of the well side, does not seem to me to be well taken.[3] One may suppose the latter replaced

[1] *Med. Times and Gaz.*, 1869. [2] *Brit. Med. Journ.*, 1869.

[3] See also Weil, *loc. cit.*

by the non-retractile emphysematous lung or even by a vacuum, and yet there would be a movement of the mediastinum in the same direction, although it would start in each case from a different position. Neither does it seem to be a correct representation to state that in pneumo-thorax the mediastinum moves toward the well side because it is no longer held by the now collapsed lung. It is true that normally the mediastinum is under the influence of the retractile power of both lungs, and is, in a sense, held by the tension of the one from following the inclination of the other to retract. But a displacement from such a cause—from failure to "hold"—could only occur in case one lung should lose a portion of its elasticity, as, for example, from emphysema. This is not the case in pneumo-thorax or, what is practically the same thing, in effusion. On the contrary, the lung remains as elastic as before, but a third factor is here introduced—namely, increased intra-pleural pressure, which, on the one hand, permits the lung to retract, and, on the other, compels the mediastinum to change its position. To say that the lung no longer holds, and therefore the mediastinum and diaphragm move, is to regard as cause and effect what are really the double effect of a third factor, the increased unilateral pressure. These dislocations are so great in even open pneumo-thorax that it is impossible to judge from them alone as to the character of the perforation in any given case.

The perforation is not always readily discoverable by simple inspection. Inflation of the lung under water will usually reveal its site by the escape of bubbles, but is of little value in determining its ante-mortem perviousness: as a rule, the perforation is found apparently open even when of many weeks' duration. It is usually minute—very rarely a tear one or more centimetres in length. It is situated oftenest in the upper lobe at its anterior margin or near the axillary line. Usually superficial, it is sometimes fistulous for a distance of one to two centimetres. Exceptionally, there are several different openings. The perforation is often partly covered and is usually surrounded by an especially thick layer of fibrin.

Pneumo-thorax is almost invariably followed by pleurisy, usually limited to the affected side, but not infrequently found upon the other as well. In all cases complicated by effusion the walls of the pleural cavity are lined with a yellowish white or grayish false membrane of varying extent and thickness; sometimes this is universal; again it may be limited to certain regions or to the site of perforation. Of 37 cases examined post-mortem by Weil, pleurisy was lacking in only 2, in neither of which the disease had lasted more than a few hours. In 32 of Weil's cases there was also effusion, and in all tubercular cases it is to be considered as one of the rarest exceptions when effusion is not present after the fourth or fifth day of the disease. The effusion is sero-fibrinous in perhaps two thirds of all cases (Netter, West, Senator, Weil); in the other third it is purulent. Putridity is surprisingly infrequent considering the usual origin of the disease. The effusion may vary in quantity from a few ounces to an amount sufficient to fill the whole pleural cavity and to completely supplant the original pneumo-thorax. The pleurisy of pneumo-thorax is always due to the invasion of germs. According to Netter, the tubercle bacillus is invariably

found in all effusions of tubercular pneumo-thorax, while the purulent forms contain also saprophytic and pyogenic bacteria; and though in a few sero-fibrinous cases no micro-organisms have been discovered by other observers, in general Netter's statement may probably be accepted. Occasionally, even in older cases of pneumo-thorax, no effusion is found, and still more exceptionally no evidences of pleurisy; these are apt to be those rarer forms of the affection such as result from simple rupture and emphysema; and they merely confirm the results of experiment,[1] which have abundantly demonstrated the usually harmless effect of the atmosphere alone upon the pleura. The tubercular and various other pathological conditions of the lungs need not here be described.

Symptoms.—A typical case of pneumo-thorax is of exceedingly acute and sudden onset. The patient, whether in robust health or already suffering from the manifestations of pulmonary tuberculosis, is suddenly seized with an intense pain, usually in the scapular region, and with excessive dyspnœa. The face is cyanotic or of a dusky pallor, the expression anxious, the forehead cold and clammy. He is unable to lie down, and either sits slightly bent forward or takes a semi-recumbent posture with body partly turned toward the affected side. There may be painful paroxysms of cough, and the voice is often husky or nearly lost. The respirations are from 40 to 60 in the minute; the pulse is weak, thready, and exceedingly rapid; the temperature is subnormal. The patient presents, in short, the symptoms of combined asphyxia and collapse. After a few hours the urine becomes thick with urates, and there is often considerable swelling of the face and extremities.

In a small proportion of cases, perhaps 8 or 10 per cent., this condition proves rapidly fatal. The opposite lung becomes œdematous, the pulse grows still weaker or imperceptible, the sensorium is clouded, the temperature fails to rise, and after a few hours, or possibly days, of agony the patient dies in collapse. Oftener the fatal termination is delayed. The severity of the initial symptoms abates, the pain diminishes, breathing becomes quieter, and by the fourth or fifth day perhaps the patient is comparatively comfortable. But with the development of pleurisy and return of the original fever his strength gradually fails; dyspnœa again increases, and death finally occurs from exhaustion some time within the first month. About half the cases survive this period, and their subsequent symptoms and course vary but little from those of the primary disease; pneumo-thorax usually increases both fever and exhaustion, but it may exist without either. In general, the symptoms of hydro- and pyo-pneumo-thorax do not differ from those of ordinary sero-fibrinous and purulent effusions. With the increase of fluid, which is often very rapid, the air apparently undergoes absorption, and it is common for a case of pneumo-thorax to soon become practically converted into a large sero-fibrinous effusion or an empyema.

But not all cases of pneumo-thorax have a violent onset. Aside from partial pneumo-thorax, the onset of which is nearly always insidious, and from those cases where perforation occurs in already moribund individuals, there are others where the initial symptoms are comparatively mild, and which are commonly described as latent. It will usually be

[1] Szupak, Wiesbaden, 1893.

found, nevertheless, that at a given time the general condition of such patients suddenly grew worse; there was a simultaneous increase in fever, pain, weakness, and dyspnœa, and this history may usually be elicited by careful questioning. But even these cases are exceptional, and it is remarkable with what definiteness most patients are able to state not only the day, but even the precise moment, of the onset of the disease.

The PHYSICAL SIGNS of pneumo-thorax are perhaps even more significant and characteristic than those of pleuritic effusion, and some of them are common to the two affections. So on inspection and mensuration we find the same immobility with even greater increase in volume of the affected side, and a corresponding displacement of the heart's impulse. The like may also be said of percussion and palpation of the heart, liver, and spleen; and for a fuller consideration of the evidences of displacement the reader is referred to the description of sero-fibrinous pleurisy (page 264). Vocal fremitus on the affected side is everywhere absent or very greatly diminished except, as in pleuritic effusion, over the compressed lung; here it is exaggerated as long as the bronchi remain pervious.

The *percussion note* in uncomplicated pneumo-thorax varies with the degree of tension of the thoracic wall. It is usually stated to be tympanitic. Weil, however, finds that tympany is not the rule, and this coincides with our own observation. We find, with Weil, that the note in general pneumo-thorax is usually loud, low pitched, and of non-tympanitic quality. Only in a certain small proportion of cases is the note plainly tympanitic, and this difference is precisely what a knowledge of the necessary conditions for tympanitic resonance would lead us to expect. A certain degree of relaxation of the walls of any closed air chamber is essential to the production of tympanitic resonance; and it is evident that in the three forms of pneumo-thorax above described —the open, closed, and valvular—the thoracic wall must present greatly varying degrees of tension, and hence also of percussion tone. The note, however, though usually non-tympanitic, invariably differs widely in pitch and intensity from the pulmonary resonance of the opposite side. It is said to be exceptionally extremely dull and muffled, almost toneless. We have never encountered this condition in general pneumo-thorax. Weil does not mention it, and West also states that it was present in none of his cases. In any event, it is always easy to determine the boundary of the distended pleura both below and at the median line in front. These boundary lines will generally show an excessive displacement of heart, mediastinum, and diaphragm, which does not differ from that of a very large effusion. It may, however, be mentioned that in left pneumo-thorax the cardiac dulness does not appear at all to the left of the perpendicular mediastinal line, while if the affection is upon the right the right perpendicular border of the præcordia may be found an inch or even more outside the left border of the sternum.

On *auscultation* the characteristic features of pneumo-thorax are a greatly diminished—almost absent—respiratory murmur over the affected side, particularly the front, and an amphoric quality of all transmitted sounds. In about 50 per cent. of all cases respiration may be faintly heard in certain regions of the chest, oftenest the axillary and posterior;

it is then distinctly amphoric, though the quality of tone is somewhat more bronchial in the neighborhood of the compressed lung. The voice sounds are also distant and amphoric, or perhaps slightly bronchial above. This same musical quality is the special characteristic of the metallic tinkle, a sound similar to that produced by dropping bits of gravel into an empty glass. Possibly, as supposed by Laennec, this sound may sometimes be caused by the dripping of fluid, but an effusion is not necessary to its production (Skoda, Behier), and it is probably oftenest due to a transmitted bronchial râle or to moisture in the perforation itself. The metallic tinkle is most frequently elicited by deep inspiration or cough. It should be remembered that the same sound may originate normally in the stomach, and be then plainly perceptible over the lower left front and axilla. Another sign of pneumothorax, thought by some to be pathognomonic, is heard on auscultatory percussion when made with two coins or with a coin as pleximeter alone; it is a distinctly musical echo which is transmitted to all parts of the affected side.

With the development of pneumo-hydro- or pneumo-pyo-thorax several very distinctive physical signs appear which are never found in simple pleurisy. As a rule, the effusion is first indicated on perhaps the sixth to the eighth day by the presence of succussion. This sign is peculiar to an air chamber containing fluid, and is a splashing sound which may be imitated by shaking a large bottle partly filled with water. It is readily detected by applying the ear to the chest wall and then giving the patient a vigorous shake; sometimes this is unnecessary, and the sound is heard at a distance or even perceived by the patient himself. It is often distinctly amphoric. Succussion is not, however, peculiar to pneumo-thorax: it may originate in the stomach or, very rarely, in a large pulmonary cavity. The explanation of the fact that succussion antedates all other evidences of fluid in pneumo-thorax is that the fluid first collects in the hollow of the depressed diaphragm, where it can produce no parietal area of flatness. But this is, as a rule, only temporary, and not long after the appearance of succussion the presence of fluid is also indicated by other physical signs which correspond in the main with those of ordinary effusion. Two of these, however, are absolutely distinctive of the presence of both fluid and air. Here there is no resisting lung to hinder the fluid from assuming immediately and under all conditions an absolute hydrostatic level. We find, therefore, first, a strictly horizontal upper line of flatness, whatever position the patient may take; and, second, an immediate change of this line of flatness with every change of position. These signs are both the more readily noticeable on percussion because of the marked contrast between the flatness of the effusion and the hyper-resonance above.

The physical signs of partial pneumo-thorax differ from the preceding only in degree, but, unfortunately, this difference is often fatal to diagnosis. Here especially the percussion note is usually dull, often even flat, and over this illy defined area, usually at a base, there is somewhat diminished voice, respiration, and fremitus. Displacement of organs can rarely be detected, and, in general, the condition is difficult of interpretation.

Diagnosis.—The peculiarly sudden and almost dramatic onset of a

typical pneumo-thorax is very characteristic, and will usually suggest at once the occurrence of perforation. So will also any marked and sudden increase of dyspnœa in an individual known to be phthisical. But the affection is often latent, and it is only on physical examination that a diagnosis can be made with certainty—a certainty, however, which is then so absolute as to be exceeded in no other condition. The combination of the three following signs is to be regarded as pathognomonic evidence of pneumo-thorax: increased or tympanitic resonance over the whole of one side; absent, feeble, or amphoric respiration over the same area; and a pronounced displacement of heart, mediastinum, and diaphragm. The only similar condition is an excessive unilateral emphysema associated with fibroid contraction of the opposite lung, the latter being often sufficient to cause a very considerable degree of displacement. Such a case has recently came under my own observation: it bore a very strong resemblance to pneumo-thorax, and it is certainly inadmissible to dismiss emphysema from further consideration on the ground usually given that it is always a bilateral affection. Sometimes a metallic tinkle or other amphoric sounds at once establish the diagnosis. But the only constant difference is in the respiration, which in emphysema is of vesicular quality, with characteristic prolonged expiration, and is never so faint as in pneumo-thorax.

Other conditions which have been mistaken for pneumo-thorax are an enormous pulmonary cavity and a certain form of subdiaphragmatic abscess. Cavities are oftenest in the upper part of the chest; partial pneumo-thorax is more likely to be at the base. A large cavity is usually in communication with the main bronchus, and presents, therefore, as a rule, normal or increased vocal fremitus and amphoric whisper; also the sign of Wintrich—a change, namely, in the pitch of tympanitic resonance according as the patient's mouth is open or closed: in pneumo-thorax all of these signs are usually wanting. Again, it is said that the peculiar effect of coin percussion is never obtained over a pulmonary cavity. In the latter also the thoracic wall is, if anything, retracted, and any displacement of organs is toward the affected side. As to subphrenic abscess, a form containing gas and recently described by Leyden as pyo-pneumo-thorax subphrenicus, may closely simulate a true pneumo-thorax. It would appear also that this condition is not so very infrequent, judging from the number of cases which have of late years been reported. It usually originates in a gastric or intestinal perforation; it is found oftenest on the left side, contains both pus and gas in varying proportion, and may, as in Leyden's case, crowd up the diaphragm as high as the third rib. The chief points of difference are—(1) History, in the subphrenic form, of a gastro-intestinal affection; (2) good vesicular respiration above the horizontal line of air or fluid; (3) but little displacement of the heart in comparison with the extent of hyper-resonance or the very pronounced displacement of the liver as shown by palpation of its free margin.

Partial pneumo-thorax presents great difficulties in diagnosis. There is often no displacement of organs or history of sudden onset, and we can only arrive at a probable conclusion by carefully weighing the sum total of all the rational and physical signs. Situated oftenest at the base and giving rise to a very pronounced dulness, or even flatness, on

percussion, it is especially apt to be mistaken for a small effusion. It may present the same curve of flatness, together with greatly diminished respiratory murmur and vocal fremitus. A valuable clue is sometimes afforded by the transmission of the whispered voice. In other cases exploratory puncture alone will establish the diagnosis.

A very rare condition which may produce all the physical signs of pneumo-thorax is diaphragmatic hernia. It is usually congenital, and, since it occurs only at intervals, the attacks of dyspnœa are intermittent. We should also expect, as in pneumo-thorax subphrenicus, to find normal vesicular respiration above.

It would be extremely desirable if the form of perforation—whether open, closed, or valvular—could be readily determined. While this is usually impossible, indications are not always wanting. The perforation is certainly valvular if puncture, with relief of dyspnœa and evident escape of gas, as shown by a hissing sound or by the movement of a feather, is followed by a rapid return of former symptoms. Complete absence of respiration over the whole affected site is generally considered a strong indication of a closed pneumo-thorax. The perforation is, on the other hand, probably still open if a loud amphoric blowing sound is heard with respiration, or if both inspiration and expiration are accompanied by a metallic sound like the bursting of a bubble (Weil): this, however, is a very rare occurrence. The degree of displacement is of little value in this connection.

The diagnosis of secondary pleurisy must depend almost entirely on the evidences of effusion. Friction is of course always absent. The horizontal line of flatness changing with the position of the patient, the presence of succussion, and the other usual signs of fluid are sufficiently characteristic to make the diagnosis exceedingly easy. In a case seen for the first time when the effusion is very large it is often impossible to determine the pre-existence of pneumo-thorax except from the history. Here, as in pleurisy, the character of the fluid exudate can only be determined by exploratory puncture.

Prognosis.—Traumatic forms are usually favorable unless complicated by infection or serious injury; effusion usually follows, but does not materially affect the prognosis. Accidental pneumo-thorax is also an extremely benign form. Cases especially of simple pneumo-thorax —those unattended by effusion, as is so common in the accidental forms of young adults—have an extremely short and favorable course. Gaillard, basing his opinion upon 22 carefully reported cases, asserts that these simple forms always recover after a duration of from ten days to two months, the confined air being rapidly absorbed by the healthy pleura. On the other hand, pneumo-thorax in the chronic emphysematous is very grave.

In the ordinary tubercular form the prognosis depends largely upon the general condition of the patient and the nature and stage of the pulmonary affection; also, to a lesser degree, upon the character of the resulting effusion. Weil's statistics of 45 cases show a mortality of 25 per cent. during the first week, 50 per cent. during the first month, and 75 per cent. during the first quarter year: 10 per cent. lived over a year, and in 2 cases there was permanent recovery. Of Saussier's 51 cases about 60 per cent. died during the first month.

It is, however, exceptional that a tolerably strong individual, with but little disease of the opposite lung, does not survive the initial attack. Favorable symptoms are but a few hours' duration of the subnormal temperature, and even some febrile reaction with the onset of pleurisy. If this fever afterward disappears at the end of two or three weeks, if the pulmonary disease is of slow evolution and not far advanced, and if the effusion is moderate in size and of sero-fibrinous character, a relatively good prognosis may then be made: there is at least the probability that the pneumo-thorax, as such, will not shorten life, and also a possibility that the tubercular process, if limited to the compressed lung, may cease to advance. Czernicki has called attention to cases of this kind, and the cessation of the tubercular process is attributed by him to the anæmia of the compressed lung. Rarely a tubercular pneumo-thorax may be uncomplicated by effusion, and the condition may continue unchanged until death; or, in still rarer instances, the air may be absorbed and the case end in complete recovery.

Purulent effusion is an exceedingly grave complication of pneumo-thorax; recovery is possible, but in most cases life can be merely prolonged. The prognosis of other forms must depend largely upon the primary disease. Double pneumo-thorax is generally fatal within a few hours or even minutes from the onset, though instances of at least temporary recovery have been reported.

Treatment.—The severe pain of the onset of pneumo-thorax invariably demands the administration of opiates, preferably subcutaneous injections of morphine: Powell also urges their necessity to counteract the effects of shock. Poultices or hot fomentations may perhaps exert a favorable influence in the same direction. The inhalation of oxygen would seem to be directly indicated where dyspnœa is of threatening intensity, and a case recently reported by Vickery[1] appeared to be greatly benefited by its use. In the majority of cases the tendency to collapse calls for energetic stimulation—alcoholics by the mouth and subcutaneously ether, camphor (1 : 10 of ether, syringeful every ten to fifteen minutes), or strychnine.

If in spite of these measures the evidences of asphyxia increase and the danger is great, an exploratory puncture should be made in the fourth to sixth interspace in front with a hypodermic needle or other fine trocar. If the perforation is valvular, as is oftenest the case in the tubercular form, the confined air will be found in a state of considerable tension, and its escape through the trocar will of course continue until atmospheric pressure prevails in the pleural cavity. But if the needle be now withdrawn, the pumping process through the valvular perforation will again begin, and intra-pleural pressure is soon the same as before. Certainly, every practitioner has noticed that puncture of a pneumo-thorax in the first few days affords at most but temporary relief. The most that can possibly be expected from any operative procedure at this stage is to produce and maintain atmospheric pressure within the pleura in such cases as exploratory puncture may have shown to be valvular. It is therefore recommended to introduce in these desperate cases a cannula *à demeure*—in other

[1] *Boston Med. and Surg. Journ.*, 1893.

words, to convert the valvular pneumo-thorax temporarily into an open one. Bouveret[1] recommends for this purpose a small silver trocar, 3 or 4 cm. in length and 3 mm. in diameter, and provided with two lateral rings at its orifice for attachment to the chest: it is inserted under the strictest aseptic precautions, and covered like a wound with a large surgical dressing. This procedure is not new, and as good authorities as Wintrich, Biermer, Unverricht, and Weil advise that a hypodermic needle be inserted and allowed to remain under similar conditions.

If the immediate effect of the perforation is survived, the question of further management is mainly that of operative treatment of the effusion. If this is so large as to threaten life, aspiration must of course be performed. It is, however, to be borne in mind that until five or six weeks have elapsed the perforation is probably but imperfectly closed, and is likely to be reopened by any diminution of intra-thoracic pressure. Unless, therefore, it be urgently demanded by the *indicatio vitalis*, no operation of a pneumo- or pneumo-hydro-thorax should be undertaken before the end of the sixth week. If at this time there is a stationary sero-fibrinous effusion of considerable size, with relatively small proportion of air, a small amount of fluid (two pints at most) should be cautiously withdrawn from time to time by aspiration.[2] In case the pleural contents are chiefly air, Weil believes that the latter should be aspirated rather than the fluid, and there seems to be no good ground of objection to this procedure. On the contrary, under such conditions the most difficult factor with which the pleura has to deal is unquestionably the air, and it is quite probable that if the latter be wholly or partially removed, the small fluid exudate will be readily absorbed. This aspiration of intra-pleural air has been done in many cases with satisfactory result.

In *pneumo-pyo-thorax* most authorities favor permanent drainage by incision as soon as the purulent nature of the effusion is discovered. Others recommend a more expectant course in all tubercular cases, and especially where there is advanced disease of the opposite lung. Senator and Fräntzel, for example, both advise small and repeated aspirations except in putrid cases, and believe that in this way life can best be prolonged. It is certainly true that operated cases of pneumo-thorax are extremely difficult to manage, and it too often happens that they do not long survive the operation. They have a great tendency to become putrid, the fistula rarely closes, and it may well be questioned whether in perhaps the majority of cases life is not shortened by the operation. Cases do, nevertheless, recover which have been treated by incision and drainage, whereas the most that can be claimed for aspiration is that it is palliative. Netter, too, calls attention to the fact that the invariable presence of saprophytic and pyogenic germs must necessarily involve constant danger of sepsis, and that on this account alone the prime indication is drainage. It is our personal conviction that the possible benefit of an operation should be denied only to those cases which are manifestly in the last stages of pulmonary tuberculosis.

[1] *Lyon médicale*, 1888. [2] For technique vide Treatment of Pleurisy, page 313.

PLEURAL ECHINOCOCCUS.

HYDATID CYST of the pleura is a rare affection, and it is still more exceptional for the disease to be primary. Of 983 cases of hydatids collected by Neisser,[1] but 17 were pleural. Maydl[2] reports 29 cases of pleural echinococcus, and of these but 8 were primary; of the secondary cases, 4 originated in the lung and 11 in the liver: one or the other of these two organs is the usual source of pleural echinococcus. The cyst is oftenest single and sterile; sometimes it contains numerous daughter cysts varying in size from a pea to a cocoanut. The mother cyst itself may be larger than a child's head. In the pleura, as elsewhere, there is an outer adventitious wall in addition to the cyst wall proper, but it is apt to be thin and poorly developed. These cysts usually grow in the direction of least resistance—namely, inward, with resulting compression of the lung and displacement of the heart and diaphragm; occasionally, for reasons difficult to understand, the growth is outward as well, and causes a local bulging of the chest wall.

The contents of the cyst may be either a clear, transparent, non-albuminous fluid or pus. In some secondary cases a pleurisy results from the rupture of the primary hepatic or other cyst into the pleura, and the daughter cysts may then be found floating freely in the serous or purulent effusion.

SYMPTOMS.—The onset is usually insidious, but it would seem that not infrequently there is at first a sharp and sudden pain. At all events, pain becomes subsequently a very prominent symptom, and is of noticeable persistency as compared with that of simple pleurisy. With the growth of the cyst there is a gradually increasing dyspnœa and a moderate cough. Physical signs become pronounced as soon as the cyst has attained to any considerable size. They are, in general, the signs of effusion—flatness, with diminution of respiration, voice sounds, and fremitus over the cyst, and over the retracted lung tympany or dulness, with perhaps bronchial respiration and the other signs of consolidation. Occasionally there is a circumscribed bulging of the chest wall, with possibly fluctuation in the intercostal spaces. Diaphragm and mediastinum are displaced in proportion to the size of the cyst. On puncture we may obtain a clear, non-albuminous fluid which the microscope may show to contain hooklets, or the fluid may be purulent and indistinguishable from that of an ordinary empyema.

The DIAGNOSIS of this affection is sometimes suggested by the constancy of pleural pain. If it is made at all, which appears to be exceptional in view of the fact that most cases have been mistaken for effusion, it must be by careful physical examination and by establishing some of the following points: a local, circumscribed bulging of the chest; in the absence of this exceptional sign a sharply defined circumscribed area of flatness, which differs from effusion in that it may be in any part of the chest and has no typical curve; on exploratory puncture, which is always to be made, a non-albuminous fluid, or, possibly, the pathognomonic hooklets, although the latter are to be expected in only a small minority of cases.

TREATMENT.—Since experience has shown that an expectant treat-

[1] Quoted by Fräntzel in *v. Ziemssen's Handbuch.* [2] Vienna, 1891.

ment of these cases is almost invariably fatal, a pleural hydatid should be operated as soon as the diagnosis becomes evident. The choice of methods is between aspiration and incision, the latter preferably with resection of a small portion of a rib. Hitherto incision has given incomparably the best results, even in the non-suppurating forms. Maydl, for example, reports 16 cases operated by puncture alone, with but 5 recoveries; incision, on the other hand, was performed in 13 cases, with recovery in all but 3. Fräntzel, nevertheless, would aspirate repeatedly in non-purulent cases before resorting to incision, and thinks the bad results of aspiration hitherto may be ascribed to imperfect technique. Since, however, incision is not a dangerous procedure, there would seem to be no good reason for delay. The operation itself and subsequent management do not differ from those of an ordinary empyema, any daughter cysts which may be present usually escaping eventually through the opening.

MALIGNANT NEW GROWTHS OF THE PLEURA.

Either sarcoma or carcinoma may invade the pleura, but, the former being quite exceptional, the following description relates chiefly to cancer.

Cancer of the pleura is in the great majority of cases secondary, either through metastasis or by direct invasion from the neighboring organs. Primary cases have, however, been reported, formerly by De la Minardière[1] and more recently by Hebb,[2] Harris,[3] and Fränkel. The usual form of pleural cancer is the encephaloid, involving, as a rule, both lung and pleura, and appearing upon the latter as isolated nodules; such growths are generally small, but occasionally they are of very considerable size, so as to cause a local bulging of the chest wall and marked displacement of organs. A peculiar form of diffuse cancerous infiltration of the pleura, to which attention was first called by Wagner in 1874, has been recently described at length by Fränkel[4] as pleural endothelioma. This variety shows an especial tendency to contraction, with a high degree of flattening or depression of the chest wall. In all forms of pleural cancer, unless the opposite surfaces of the membrane are firmly adherent, there develops sooner or later an effusion into the pleural cavity, which is oftenest a simple transudation, and in about two thirds of all cases is more or less hemorrhagic.

Symptoms.—The rational signs are neither constant nor characteristic. There may be dull persistent pain in the side, but, aside from this and from the progressive weakness and dyspnœa which would naturally accompany any considerable growth within the thorax, the symptoms are mainly those of the complicating pleurisy and of the primary disease. On physical examination a varying degree of cachexia may be noticeable. The glands of the neck and axilla are not infrequently found enlarged. In some cases of large nodular growth in the pleura there may be a circumscribed bulging of the chest; in others there is a marked

[1] *Thèse de Paris*, 1875. [2] *Lancet*, 1893. [3] *Brit. Med. Journ.*, 1892.
[4] *Berliner klin. Woch.*, 1892.

retraction of the lower lateral and posterior aspects of the thorax, the result of a rapid shrinking process such as attends the more diffuse and fibrous forms of the disease. The signs on auscultation and percussion must evidently vary exceedingly with the size and seat of the growth, and especially with the amount of accompanying fluid. Here it need only be said that a large uncomplicated pleural growth may cause very considerable displacement of the diaphragm and mediastinum, and an atypical area of flatness, over which there is great diminution of voice, respiration, and fremitus. In the endothelial form of diffuse fibroid thickening of the whole pleura there may be, even without much fluid, dulness or flatness with absence of respiration over the whole affected side. Fluid, however, is usually present in all varieties of pleural cancer, and greatly obscures the signs of the growth itself.

Our most valuable source of evidence in malignant disease lies in exploratory puncture. The important factors in diagnosis are thus laid down by Fränkel:[1] (1) A deep red color of the fluid, almost like that of venous blood. A simply hemorrhagic fluid is in no way characteristic of cancer. (2) The discovery on microscopical examination of small particles of the growth itself which show its organic structure. These are of course encountered only when the process is destructive. Groups merely of polymorphous cells may be found under other conditions than cancer, and are not therefore sufficiently diagnostic. (3) The presence of considerable fat, either free as a chylous fluid or enclosed in epithelial cells. If the latter contain vacuoles, are polymorphous or polyhedric in contour, or are so stuffed with fat globules as to present the mulberry appearance described by Quincke, the evidence is very much strengthened: fat alone may be found also in a tuberculous effusion.

Taken together, and particularly in connection with a rapidly developing retraction of the chest wall, these signs afford very strong evidence of pleural cancer; if with advanced age there are also cachexia and enlargement of the cervical and axillary glands, the diagnosis may be made with considerable confidence. In the absence of these conditions, especially of a hemorrhagic effusion, it is extremely difficult and often impossible to distinguish a malignant growth from a pulmonary consolidation or an echinococcus. It is to be borne in mind that the mere negative value of exploratory puncture is not very great; the fluid of cancerous pleurisy does not necessarily contain blood or present any of the characteristic features above given.

Prognosis and Treatment.—Like inoperable cancer elsewhere, pleural cancer has an inevitably fatal course in from six to eighteen months. Sometimes life is abruptly terminated by an intra-pleural hemorrhage, or, in the very old, the complicating pleurisy may prove the immediate cause of death. The treatment can only be palliative and sustaining. Temporary relief can often be afforded by aspiration of the accompanying effusion, although it will, as a rule, be rapidly reproduced, and the operation must be frequently repeated.

[1] Loc cit.

DISEASES OF THE CIRCULATORY SYSTEM AND THE MEDIASTINUM.

DISEASES OF THE CIRCULATORY SYSTEM AND THE MEDIASTINUM.

PHYSICAL SIGNS OF CARDIAC DISEASE.

By ELBRIDGE G. CUTLER, M. D.

Structure of the Heart.

Anatomy.—The heart, enclosed in the pericardial sac, lies in an oblique plane extending from the right side downward and forward toward the left side. It is situated partly behind the sternum and partly behind the right and left costal cartilages. Its highest point, the upper border of the left auricle, corresponds to a line connecting the lower borders of the sternal insertion of the second pair of ribs. Its lowest point is the middle of the upper border of the sixth left costal cartilage. The heart extends eight or nine centimetres to the left and four or five centimetres to the right of the middle line of the sternum. In relation to the chest wall we distinguish in the heart a right, a left, and a lower border.

The right border is formed by the right auricle, and runs, in a line curving outward two or three centimetres beyond the right edge of the sternum, from the middle of the second right intercostal space to behind the sternal end of the fifth right costal cartilage.

The left border runs in a convex curve from the second left intercostal space downward and outward to unite with the left end of the lower border at the apex of the heart.

The lower border is formed by the right ventricle, and extends from the sternal end of the fifth right costal cartilage in a slightly descending line to the fifth left intercostal space, where it meets with the left border in the mammillary line or a trifle inside it.

By far the greater portion of the heart is covered by lung; only a segment of the organ, belonging exclusively to the right ventricle, lies directly against the chest wall. The boundaries of this area, which can be determined only by percussion and auscultation, will be fully described later on.

As in suspected pulmonary disease, so also in cardiac cases, we begin our investigation with inspection of the naked chest, followed successively by palpation, percussion, and auscultation.

The section of the chest which overlies the heart is called the præcordia.

Examination of the Heart.

Inspection.—The person to be examined must either stand, sit, or recline in such a position that the light, preferably daylight, shall fall evenly on both sides of the front of the chest, entirely denuded of clothing. He should not, just before the examination, have undergone any considerable mental or bodily excitement. There is no difference in the two sides normally in the arching of the wall or thickness of the layers of skin and muscle.

We begin the examination of the heart by inspecting the cardiac movements, and, although what follows chiefly concerns what is seen with the eye, it is confirmed and increased by the touch. In the healthy person these movements are of two kinds—either (1) a diffuse trembling of the entire cardiac area or a greater part of it, the impulse of the heart; or (2) a circumscribed lifting of a small section, the apex beat. The latter is more frequently observed.

Apex Beat.—The apex beat in adults is seen in the fifth left intercostal space, just within the mammillary line. In a very short thorax it may appear in the fourth intercostal space. In early childhood the apex beat is found to be higher, as a rule. Up to the fourth year in most cases it is situated in the fourth intercostal space; up to about the sixth year it is just as frequently in the fourth interspace as in the fifth; but from the seventh year on it is more frequently found to lie in the fifth intercostal space. Up to the sixth year it is for the most part found outside or in the mammillary line, and from the seventh year, in the majority of cases, and from the thirteenth year almost exclusively, inside the same.

The apex beat is not visible in all persons. It is not rarely absent in fat people, especially women, and in a short thorax with small interspaces. Where it lies behind the sixth rib instead of the fifth intercostal space, as sometimes happens, it is also lost to view. The thinner and more flexible the thorax the more evident is the apex beat; hence in children it is most marked.

Under normal conditions the heart is subject to passive changes of position accompanying the altered posture of the body. It sinks from three to six centimetres to the left, or from one and a half to three centimetres to the right, according as the body is lying on the left or the right side. The heart retracts slightly also if the sitting or upright position is exchanged for the horizontal, a slight change being produced in the cardiac impulse. The heart also follows the movements of the diaphragm, being dragged down by an inspiration and raised by an expiration. The difference of level produced in this way in the heart's position amounts to about the breadth of an intercostal space.

In diseased conditions we may notice the following abnormality of the anterior thoracic wall: If the volume of the heart is increased to a considerable degree by hypertrophy or dilatation, or both, or if the pericardium is extensively swollen by a large exudation into it, we may observe a protrusion of the region of the left side which lies over the heart—namely, the part between the left margin of the sternum and the left nipple from the third rib down to the seventh. This occurs most frequently in early life, while the chest is still flexible. In

exceptional cases, particularly in large pericardial exudations, this protrusion may extend as far as the sternum or even beyond to the right side, causing retraction of the left lung. Such a protrusion may also occur from curvature of the spine where the middle dorsal vertebræ are convexly curved to the left, and must be carefully differentiated from the above conditions. It is only in cases of extraordinarily large pericardial exudations that we see the intercostal spaces bulging forward.

Pathologically, the apex beat may either be wanting, be increased in force, or occur at an abnormal point. Its disappearance may be due (1) to emphysema of the lung where the apex is overlaid by distended lung, which exceptionally may be more developed on the left than on the right side. (2) A pericardial exudation or a pneumo-pericardium, very rare, may give rise to the same disappearance. (3) A pleural exudation may overlie the apex, or a pneumo-thorax of the left side. (4) Weakening of the heart through disturbance of the nutrition, as anæmia, obesity, diabetes mellitus, gastric diseases, may cause it. (5) Also unusual thickness of the thoracic wall from anasarca or obesity.

Increase in the apex beat may be seen in otherwise normal conditions of the heart, in increased cardiac activity due to psychic excitement during the examination, in nervous heart beat, and after bodily exertion. In a feeble heart the same appearance may follow from its increased irritability. In increase of the apex beat the latter is, moreover, seen over a greater area; it is enlarged. These are all temporary conditions of activity; a permanent increase of the force of the apex beat always indicates disease.

This increase may occur in a heart of normal shape and size or one which deviates but slightly from the normal, and is seen especially in permanently increased cardiac activity, as in the neuroses, particularly Graves' disease, and in fever not yet advanced to a condition of considerable cardiac weakness. An endocarditis at its very beginning may be indicated by an increased apex beat before a valvular lesion or hypertrophy of a ventricle has developed. This is true of acute myocarditis. Finally, the apex beat may appear to be increased in a thorax of abnormal shape, as in the rachitic pigeon breast, because from lack of room the apex is pressed deeper into the intercostal space than normally.

It is, however, hypertrophy of a portion or the whole of the heart which especially causes a protracted increase in force and area of the apex beat. It is therefore observed most frequently in valvular lesions. In exclusive or predominating hypertrophy of the right ventricle, provided it is not dependent on marked emphysema of the lung, we see the increased and broadened apex beat in the fifth intercostal space, usually in the mammillary line or at the same time just within and without the same. (Where the lung overlying the heart is markedly emphysematous the apex beat is either not seen at all or is very feeble, and on account of the low position of the diaphragm appears in the sixth intercostal space inside the mammillary line. On the other hand, we frequently see in such a case a systolic protrusion in the epigastrium not dependent on the cardiac apex.) In hypertrophy exclusively of the left ventricle the apex beat is outside the mammillary line, as a rule in the sixth intercostal space; less frequently—in slight degrees of hypertrophy and in young children—in the fifth; not so very rarely—in extreme hyper-

trophy and dilatation—in the seventh intercostal space even. This is the case when the right ventricle as well as the left is dilated and hypertrophied, as, for example, in mitral regurgitation. The apex beat here appears very markedly broadened, so that the protrusion can be seen also within the mammillary line. We have here, besides the increase in strength, a displacement of the apex beat outward and downward, which is especially characteristic of hypertrophy of the left ventricle. The accidental discovery of an outward and downward displacement of the apex beat may be the first thing which leads us to an examination of the urine and the discovery of a cirrhotic kidney, or may draw attention to an aneurysm of the aorta hitherto without symptoms.

Other displacements of the apex beat are often of great value in diagnosticating the cause of the disease or of estimating its degree. The displacement of the apex beat outside the mammillary line to the left in a right-sided pleural exudation shows us instantly that the fluid has reached a certain amount. The greater the increase of fluid the more considerable becomes the cardiac displacement. In such cases we find the apex beat frequently three to five centimetres outside the mammillary line in the fifth intercostal space, or in great exudations even in the fourth interspace, when it may be in the axillary line. (The apex of the heart is raised as a necessary consequence of the sinking of the base caused by traction of the vena cava inferior following the downward displacement of the right half of the diaphragm and of the liver, and aided by the combined action of the upward tilting of the left lobe of the liver and the displacement of the air-containing abdominal organs (C. Bartels).)

In left-sided pleural exudations the apex beat moves toward the right or wholly disappears, the cardiac apex then being covered by the exudation. A considerable pneumo-thorax with marked tension of the air in the pleural sac has the same effect as a pleuritic exudation of the same side. An impulse of the heart is seen on the right border of the sternum between the third and fifth ribs, and corresponds with the base, not the apex, the heart in dislocation not altering its parallels.

Similar displacements of the apex beat to the right or left are caused by shrinkage of a lung on the corresponding side. In right-sided shrinking of the lung the apex beat, as a rule, is in the fifth, or in young children in the fourth, intercostal space, right side. In very marked shrinkage it may wholly disappear. In left-sided shrinking, according to the degree, we find the apex beat either in the fifth or fourth intercostal space, always outside the mammillary line—in great degrees of shrinking even in the middle axillary line, and then always in the fourth intercostal space.

With a high position of the diaphragm the apex beat, together with the whole heart, is dislocated upward, and appears in the fourth interspace, usually inside the mammillary line.

In inversion of the viscera of course we find the apex beat on the right of the sternum in the fifth interspace, just within the mammillary line, and any displacements which it suffers as the result of cardiac disease, pleurisy, or shrinkage of lung follow the same general laws as when the heart is in the normal position. If the condition is one simply of *dexiocardia*, however, the altered position of the great vessels with

relation to each other, and the relation of the inferior vena cava to the liver, will be such as to create confusion and render a proper estimation of the condition impossible during life.

A diffuse cardiac impulse is seen in retraction of the borders of the lungs, which is due to superficial breathing, as in anæmic, feeble, and bedridden patients, or to contraction of the lung or enlarged heart, whereas in a large pericardial exudation which pushes back the lungs an impulse is not seen any more than is the apex beat. The cardiac impulse in such cases appears in the region of the third, fourth, and fifth intercostal spaces between the left edge of the sternum and the place of the apex beat. In hypertrophy of the left ventricle or displacement of the heart to the left it may extend beyond the mammillary line. If the ribs and cartilages are elastic, both they and the lower end of the sternum may be distinctly lifted in strong pulsation. Sometimes the movement is wavelike, a sinking in occurring in the third and fourth intercostal spaces close to the edge of the sternum at the same instant that a bulging follows in the fourth and fifth spaces a little farther off from the sternum.

Where the heart is drawn or pushed to the right there is often a cardiac impulse to the right of the sternum, usually in the fourth and fifth intercostal spaces, appearing as a simple systolic protrusion, and always near the sternal edge.

A systolic retraction of the apex beat is sometimes seen, which is usually dependent on a tolerably strong adhesion of the heart to the pericardium, and often of the latter to its surroundings (diaphragm, anterior thoracic wall, pleura, or vertebræ). Abnormal folds in the posterior portion of the pericardium may also cause it.

Pulsation.—Epigastric pulsation may be seen in thin people, depending on the movements of the abdominal aorta. Such pulsations are very marked where the heart acts vigorously, as in excitement or in neurosis, or when the stomach is full, or where there are tumors in front of the aorta, as a degenerated pancreas, swollen lymph glands, or cancer of the stomach. Aneurysm of the abdominal aorta high up may give rise to the same thing.

Pulsation in the hepatic vein may give rise to the epigastric pulsation also.

Hypertrophy of the right ventricle following great emphysema may cause epigastric pulsation, the apex beat being concealed by overlying lung.

Pulsations in abnormal places may be due to aortic aneurysms, as in aneurysm of the ascending aorta, where the protrusion appears in the first and second intercostal spaces to the right of the sternum. Or an aneurysm of the arch may give pulsation in the jugulum in the left first intercostal space close to the sternum.

Sometimes in pleuritic exudations an extensive systolic pulsation may be seen in several intercostal spaces, depending on a participation of the fluid with the cardiac movements. This is most frequently seen in left-sided exudation. It is favored by relaxation of the intercostal muscles from serous imbibition in great tension of the exudate. In empyema necessitatis the pulsation may be confined to the prominent point. (See Pulsating Pleurisy, p. 288.)

Rhythmical movements may be observed in the distended jugular veins, sometimes depending on the respiratory excursions, at others on the cardiac pulsations. Even when the veins are normally full the expiration may check their emptying when it is somewhat prolonged, as in coughing, straining, and so forth, the pressure inside the chest driving the blood back through the vena cava and innominate up to the valves of the common jugular and closing them, thus causing a collection of the blood above. If the cervical veins are permanently overfilled, an expiration of normal duration may cause an observable distention, which disappears on inspiration, so that there is a rhythmical increase and diminution of the swelling during the ordinary respiratory movements. This appearance may be much increased by dyspnœa. The opposite condition, inspiratory increase and expiratory diminution of the size of the jugular, may be observed in substernal goitre or mediastinal tumors, which on inspiration are made to press on the venæ innominatæ by the lifting of the upper part of the thorax, or the traction of adhesions in a cicatricial mediastino-pericarditis may cause narrowing of these veins or of the vena cava superior.

If the cervical veins are permanently dilated and overfilled, there may be a presystolic swelling and a systolic collapse from the auricular contraction. The wave of blood thrown into the superior vena cava and innominatæ by the auricular contraction closes the valves in the common jugular vein, and causes a temporary passive congestion above them. The intumescence below the valves normally concealed behind the sterno-clavicular joint, and designated as the *bulbus venæ jugularis*, is brought into sight in the groove between the two heads of the sterno-cleido-mastoid muscles as the result of the dilatation and displacement. In this bulbus we then have a presystolic pulse, but above it only a presystolic interruption of the blood current corresponding to the pulse mentioned, a swelling of the same, and instantly after it the systolic collapse of the vein follows. We may see precisely the same thing in the external jugulars, though always in a less marked degree, owing to the more remote point of emptying of the vein into the subclavian. If in such cases the venous valves are insufficient as the result of extreme dilatation, the blood wave may be thrown back by the contracting auricle and reach the body of the vein, and cause here a presystolic pulse.

If, now, there is tricuspid insufficiency due to extreme dilatation of the right ventricle or to congenital causes, there is seen not only during the contraction of the right auricle, but also in systole of the right ventricle, a blood wave thrown back into the venæ cavæ and innominatæ, and appearing at the bulbus (and in insufficiency of the venous valves in the common jugular as well) as a presystolic and also a systolic pulse.

These systolic and presystolic bulbar and jugular pulses appear more frequently on the right than on the left side ; if they occur on both sides, they are earlier and more distinct on the right.

If the conditions which give rise to a jugular pulse are present, a systolic pulse in the liver (hepatic vein) may also be observed, though far less frequently. As a rule, this is seen only in tricuspid insufficiency (usually relative insufficiency). In rare cases a presystolic pulse is also detected, but chiefly through the aid of palpation and sphyg-

mographic tracings. The hepatic pulse is a diffuse symmetrical systolic (at times also diastolic) enlargement of that part of the liver which extends below the edge of the ribs. It occurs especially in the epigastrium, more particularly to the right of the median line.

A pulse is very rarely seen in veins more distant from the heart, as, for example, the anterior jugulars, the thyroids, the external thoracic, the veins of the arm, frontal and temporal veins. Still more rarely a pulse in the veins of the lower extremities has been observed in tricuspid regurgitation: in such cases there must be not only varicosity of the crural veins, but insufficiency of the valves as well.

The name *progressive venous pulse* was given by Quincke to a pulsation of the veins of the back of the hand and foot which comes from a continuation of the arterial pulse through the capillaries into the veins. It has the same significance as the capillary pulse. The same thing is occasionally seen in insufficiency of the aortic valves.

PALPATION.—Palpation confirms and completes the results of inspection, and sometimes very materially adds to them. It is consequently used at the same time as inspection.

In palpation the finger is placed over the region of the heart and experiences a slight elevation. This may be confined to the apex and be covered by one or two finger-tips, or it may be found over a larger area. It must be remembered, however, that the force of the impulse varies according to the extent to which the lung covers the apex of the heart, according as the patient breathes superficially or deeply, and, lastly, whether he is excited or not. It frequently happens that by means of the touch we can find the cardiac impulse where inspection has failed to reveal it. In many cases of emphysema the lung overlying the heart renders the impulse imperceptible to the eye, while the finger feels it readily. On the other hand, exudation into the pericardium may render the impulse imperceptible both to the eye and finger. Palpation enables us to estimate the intensity of the cardiac pulsation rather more exactly than does inspection. If the left ventricle is hypertrophied, the finger appreciates an abnormal resistance at the apex, but if the impulse is so powerful as to shake the anterior chest wall, palpation is not necessary to disclose it. Palpation is, however, of great value in cases of secondary hypertrophy of the left ventricle due to contracted kidney or sclerosis of the aorta where no special changes are yet observable in the position or extent of the cardiac impulse. Palpation is also important in enabling us to establish a decrease in the power of a once normal heart, depending on fatty degeneration or pericardial exudation.

The closing of the valves is frequently perceived by palpation. Traube, who first called attention to this fact, did not consider a systolic valve impulse as of any special diagnostic significance. It is seen in many healthy persons in whom an unusually large portion of the heart is covered by the anterior edge of the left lung. Where there is complete absence of the apex beat we may find, as Traube says, "a trembling at the time of the ventricular systole in the region of the third to the sixth costal cartilage, and at the lower portion of the sternum," which can be produced by nothing else than the vibrations of the mitral and tricuspid valves. Eichhorst says a diastolic valve impulse may be felt just as frequently. It is a short, distinct diastolic

stroke which appears to come from deep in. It is usually found most distinctly over the sternum at the level of the second and third costal cartilages, but not infrequently it is propagated downward a little. An excited or energetic heart's action is not necessary to produce it, nor need there be any change in the course of the edges of the lungs. It has no diagnostic significance.

We may have a localized diastolic valve impulse in the following pathological conditions: (1) when the middle portion of the edge of the left lung which covers the first part of the pulmonary artery, and separates it from the chest wall, is infiltrated, and therefore airless; or (2) when it is retracted outward so far that the pulmonary artery lies directly against the chest wall. The impulse is oftenest seen in the second left intercostal space close to the sternum, and comes from the semilunar valves of the pulmonary artery. A visible pulsation of the pulmonary artery is usually connected with it.

When the right ventricle meets with considerable resistance from lesions of the mitral valve or chronic pulmonary diseases, there is an increase in impulse of the pulmonary valves. If the index finger of one hand is placed in the second left intercostal space close to the edge of the sternum, while the other hand is placed at the apex beat, there is perceived the alternate systolic lifting of the apex beat and the diastolic short stroke of the pulmonary artery. A diastolic valve impulse is much more infrequently observed in the second right intercostal space near the sternal edge, dependent on the semilunar valve of the aorta. The cause lies in the abnormal resistance which the left ventricle meets in its action.

Pathological changes in the heart frequently give rise to palpable murmurs, called thrills, though usually murmurs are chiefly determined by auscultation. The palpable like the audible murmurs are divided into endocardial and exocardial murmurs. Usually exocardial and endocardial murmurs can be differentiated by the sense of touch. The palpable exocardial murmurs give the impression of rubbing, scratching, scraping, and are characterized by intermittency, while the endocardial palpable murmurs are continuous, since they are produced by blood currents and feel like the purring of a cat or the vibration of a cello string. The differential diagnosis is facilitated when the murmur can only be felt by firm pressure in an intercostal space, as this indicates pericardial roughening. The time of the murmur is of importance. Endocardial murmurs are always exactly either presystolic, systolic, or diastolic, while exocardial murmurs are neither wholly systolic nor diastolic, but drag, so that sometimes they seem more the one, sometimes more the other. The feeling disappears on deep inspiration frequently, because the left lung interposes between the thoracic wall and heart. Laennec called the palpable endocardial murmur frémissement cataire, and the palpable exocardial murmur, frottement.

As a rule, the frémissement—or, as we call it, thrill—accompanies a loud endocardial murmur, so that it sometimes happens that it disappears while the heart acts quietly. Let the patient get physically or mentally roused, however, or take frequent long breaths, or move round rapidly, or change his position quickly from a lying to a sitting posture, and it returns again.

It is more especially with organic valvular murmurs that the thrill is felt, though the same has been observed, though rarely, with inorganic valvular murmurs. Experience teaches us that thrill is less frequent over some valves than over others. Thrill at the apex depends on mitral disease, and is more frequent in stenosis than in insufficiency; accordingly, a diastolic or presystolic thrill is oftener felt than a systolic thrill. A presystolic thrill furthermore is frequently more distinct at the beginning and end of its time of duration than in the middle.

A very distinct thrill is often found in narrowing of the aortic orifice. We feel it in the second right intercostal space and over the neighboring section of sternum. Diastolic thrill in insufficiency of the aortic valves is not often felt; its greatest intensity is usually over the body of the sternum.

Thrills depending on disease of the valves of the right side of the heart are great rarities. Such are sometimes found in the second left intercostal space over the pulmonary artery and over the lower end of the sternum in disease of the tricuspid valve.

A very distinct and widespread thrill has been felt where there was abnormal communication between the two sides of the heart.

The palpable pericardial friction rub occurs, we might almost say, only when the layers of the pericardium have become rough and uneven through inflammation and exudation of fibrin on their otherwise normally smooth surfaces. It is not necessary that both surfaces should be diseased; one may be normal. Furthermore, the distinctness of the rub and the loudness of the sound on auscultation are no criteria of the extent of the diseased process; they depend rather on the location of the disease. Sometimes slight hemorrhage is sufficient to cause a pericardial friction, and simple dryness has been asserted to cause it also.

We most frequently feel the pericardial friction near the left edge of the sternum.

Percussion.—Percussion of the heart is best practised with the fingers in the same way as already described under Percussion of the Lungs, the patient either lying on the back with the upper part of the body a little raised or in the sitting posture. In the latter case the examiner sits in front of the patient, or in determining the lateral boundaries of the cardiac dulness often stands behind or on one side of him. With the patient lying down one stands either on the right or left as is most convenient in determining the boundary of that side.

The position of the apex beat is first determined by inspection and palpation where possible, and the point marked with a cross. If it is neither visible nor palpable, we proceed directly to percussion.

The portion of heart uncovered by lung is bounded on the right by the middle border of the right lung, which during life is near the left edge of the sternum and runs almost parallel with it to about the level of the fifth costal cartilage, where it curves into the lower border of the lung; above and to the left it is bounded by the incisura cardiaca of the upper lobe of the left lung, while the tongue-like end, as a rule, covers the apex of the heart. The lower border is formed by the lower edge of the heart and joins the area of liver dulness.

We have then an area of flatness and one of relative dulness of the heart. The area of flatness is not absolutely so, but with gentle percus-

sion, which should always be used in cardiac percussion in this locality, it is sufficiently near it.

The shape of the area of cardiac flatness is an irregular quadrangle, the right and lower borders of which are straight and form a right angle with each other, while the upper border is straight and nearly horizontal, with its outer end at a little lower level than the sternal end. The outer border is slightly curved. The *right border* of the flat area is found to be at the left edge of the sternum, for, though the right lung does not reach quite to the sternal edge, the pulmonic resonance is conducted thus far on percussion.

The *upper border* is found by percussing gently in straight lines from the second intercostal space vertically downward parallel to the sternum. In the ordinary adult between the ages of twenty and fifty we come suddenly upon a line at the upper border or middle of the fourth costal cartilage where the loud pulmonary resonance changes to the flat cardiac sound. The *left border* is found by beginning our percussion in the mammillary line at the fourth rib, and percussing toward the sternum and in horizontal lines parallel to this down to the apex. The left border is not always a straight line, but is often curved, and it may form a curve instead of a broad angle with the upper border.

The *lower border* cannot be outlined except when with gentle percussion the tympanitic resonance of the stomach is conducted through the left lobe of the liver. As a rule, we are forced to draw an imaginary line from the edge of the sternum outward to the apex. Sometimes the heart extends farther to the left than does the left lobe of the liver, in which case the outer end of the lower border can be easily determined by percussion, and is then found to be almost exactly at the same level as is the lower border of the lung in the right mammillary line.

To determine the area of relative dulness of the heart we employ stronger percussion than for the absolute flatness, but the directions in which we percuss are the same.

The *upper border* of relative dulness is usually found at the upper edge of the third left costal cartilage. It runs usually without any sharp angle in a gentle sweep into the *left or outer border*, which begins in the third or fourth left intercostal space or fourth rib, and ends in the fifth intercostal space just about one centimetre outside the mammillary line at the upper border of the sixth left rib. The *right border* of relative dulness is found by percussing toward the sternum from midway between the right edge of the sternum and the corresponding parasternal line about four centimetres above the lower border of the lung. It curves outward in a convex line to the upper edge of the sternal insertion of the fourth right costal cartilage, and ends at the upper edge of the sternal insertion of the sixth left costal cartilage.

To determine the *breadth* of the relative dulness of the heart we measure the distance from the outermost limit of the left border in the fifth intercostal space to that of the right border at the same level. To determine the height we measure the distance, in a vertical line close to the left edge of the sternum, from the upper border to the point where liver and heart dulness joins. The breadth of relative dulness in the adult is fifteen and a half to sixteen and a half centimetres, and the height eleven to twelve centimetres.

The breadth of the absolute flatness in adults of middle age is in normal conditions, as a rule, the same as the height, and averages five to six centimetres.

In children up to about the twelfth year the upper border of absolute cardiac flatness is, as a rule, somewhat higher than in middle-aged adults—namely, at the third intercostal space. The left border usually lies somewhat farther out, also corresponding to the position of the apex. The whole area of cardiac flatness, therefore, appears relatively higher and broader.

In aged persons, on the other hand, the upper border stands at a lower level, about in the fourth intercostal space or on the fifth rib, and at the same time the left border moves toward the sternum somewhat though not in the same proportion. In the aged the entire area of absolute flatness is materially diminished, and especially in height. This change with increasing years is the result of atrophy and dilatation of the lungs.

The relative cardiac dulness appears to be broader in children than in grown people. The right border is farther to the right, even reaches in very young children the parasternal line; the upper border is in the second intercostal space on the left; the left border extends somewhat farther beyond the mammillary line. On the other hand, the lower border in children up to six years of age stands higher than in adults, corresponding to the position of the apex beat, and is at the lower or even upper border of the fifth rib. It thus appears that the height of both the absolute flatness and relative dulness is no greater in the child than in the adult in proportion to the size of the thorax, but the breadth is a little greater.

The absolute cardiac flatness suffers certain changes in shape through the respiratory movements. It becomes smaller in inspiration and larger in expiration, but in quiet breathing these variations are slight. With a deep inspiration the upper border descends two to two and a half centimetres, while the left border moves one and three quarters centimetres to the right, the right border remaining unchanged. The displacements which follow expiration are of course the opposite of the inspiratory ones. They amount to about two centimetres outward and upward, and are not much increased when the expiration is deep, the right border remaining unchanged. The same changes on respiration are also found in the relative cardiac dulness, and they are about the same in kind and extent as in the absolute flatness.

Changes in the position of the body to the right or left lateral decubitus give rise to a so-called passive mobility of the cardiac flatness and dulness, which are of theoretical rather than clinical interest, since we usually examine the patient in either the dorsal or upright position. These changes do not vary much from those depending on the respiratory movements, and need no further description.

Pathological Conditions.—Changes in the shape and size of the absolute cardiac flatness may occur independently of diseased conditions of the heart, as well as in connection with them. In the latter case there are also alterations in the relative dulness which will be considered at the same time.

(*A*) It is not uncommon to find with the lungs in an otherwise normal

condition *an enlargement of the absolute cardiac flatness* in persons who have breathed superficially for a long time. Such a condition is especially seen in patients who have been in bed a long time on account of severe illness or in chlorotic and anæmic individuals. In such cases the absolute flatness may be enlarged in all directions. We constantly find the upper border higher than normal, about in the third intercostal space, and the left border pushed outward. It is not rare to find also an enlargement toward the right at the lower end of the right border of the sternum. The superficial breathing may also be due to pain caused by the respiratory movements, as in the pleurisy of pneumonia, dry pleurisy, more infrequently dry pericarditis, or even peritonitis. A high position of the diaphragm following pregnancy, abdominal tumors, great ascites, and peritonitis with exudation and meteorism, has been observed to cause enlargement of the absolute flatness. In such cases it is the encroachment on the space in the thorax which allows the lungs to retract and uncovers the heart.

Lastly, an enlargement of the absolute flatness, the lungs being otherwise normal, may occur from an increased collection of fat in the anterior mediastinum, which pushes aside the lungs, especially the right, as is seen in ordinary obesity. In such cases there is also not infrequently a dilatation of the heart as well, as is shown by an increase in the relative dulness, and which may reach a high degree. The dilatation is not necessarily connected with the condition mentioned, and the increased absolute flatness may remain when the dilatation has been reduced by appropriate treatment.

(*B*) Changes in the absolute flatness may be due to pathological conditions of the lungs and pleuræ, and may consist of diminution, increase, or displacement of the same.

(1) Diminution of the absolute cardiac flatness may temporarily result from deep inspirations, as in acute emphysema, or it may be permanent, as in interstitial emphysema. It is the left lung almost wholly which causes the diminution of the flat area here, and if there is contraction of the right lung, emphysematous dilatation of the left may be the sole cause. If the emphysema is of high degree, the absolute flatness may be almost or wholly absent; for the heart is so nearly covered by distended lung that the cartilages of the ribs conduct the pulmonary resonance on percussion over the small uncovered portion, and the pulmonary resonance extends down to the hepatic flatness or very nearly to it.

The relative cardiac dulness can be demonstrated on the left of the sternum even in the highest degrees of emphysema, though it is somewhat smaller in extent on account of the increase in thickness of the layer of lung covering the heart. We often find the extreme left border, therefore, in the mammillary line or even a trifle inside it, and the upper border about on the upper edge of the fourth costal cartilage. We must remember that the lower position of the upper border here corresponds to a lower position of the diaphragm and of the whole heart.

In extreme emphysema it is difficult to determine the relative cardiac dulness on the right of the sternum. But with practice we can succeed, and can frequently demonstrate an enlargement of the relative dulness

to the right corresponding to hypertrophy of the ventricle and dilatation of the auricle. It is to be borne in mind that this boundary found will correspond less exactly with the true one than in the normal condition of the lungs.

(2) An increase in the absolute flatness is observed in contraction of the left lung. If the contraction affects the upper lobe, the flatness appears increased upward and to the left, but its boundary can be determined only when the adjoining lung still contains air. If the contraction is great, the apex beat is lifted upward and outward and the lower border of flatness is displaced upward correspondingly.

In contraction of the right lung of moderate degree the absolute flatness on the left of the sternum is diminished, the right border is more or less distant from the left edge of the sternum, and the left border of relative dulness inside the left mammillary line. In great contraction of the right lung the absolute flatness may almost entirely disappear from the left of the sternum, and a greater part of it be found at the lower end of the sternum and to its right, while the left border of relative dulness, as well as the apex beat if it still be seen and felt, will be met at a short distance from the left border of the sternum. The right border of absolute flatness and of relative dulness in such cases can only be determined if the neighboring lung contains air.

(*C*) It is impossible to map out the absolute flatness and relative cardiac dulness when a flat region depending on pathological processes immediately joins them. In complete consolidation of the upper lobe of the left lung from pneumonia the upper and left borders of cardiac flatness and dulness cannot be determined, and with the same process in the right middle lobe the right border of these areas also cannot be found. The same is true in contracted lung on either side if the pulmonary tissue immediately joining the heart contains no air. In advanced phthisis of the left lung it is the upper border and the upper portion of the left border which cannot be determined. On the right side phthisis seldom leads to so complete a condensation of the middle lobe as to drive out the air from the anterior edge and thus conceal the right border of absolute flatness.

Sacculated pleural exudations according to their position may render the determination of the one or other boundary of the cardiac flatness or dulness impossible. The same is true of tumors in the neighborhood of the heart. Peripleuritic abscesses, finally, may conceal the whole or a part of the cardiac dulness and flatness on one side of the sternum.

Collections of fluid or gas in a pleural sac may decidedly either influence the cardiac dulness by rendering a determination of the boundaries impossible, or by crowding on the heart may cause a displacement of the same. But such collections must be considerable to influence the cardiac percussion in the upright or dorsal position. In medium-sized exudations of the left side the left border of cardiac flatness, as well as of relative dulness, disappears first, the dull heart sound merging into that of the fluid. As the exudation increases the possibility of determining the upper border diminishes, so that finally the whole left side of the thorax is flat, and the cardiac flatness cannot be distinguished from that of the fluid. At the same time, there is displacement of the heart, the cardiac flatness being found at the sternum and to the right of it, while

the right border of relative dulness, with increase of the displacement, moves farther and farther to the right, till it finally may reach the mammillary line. In pleuritic exudations of the right side the right borders of flatness and dulness disappear gradually as the exudation increases, till they finally blend. When the exudation is great the heart is displaced to the left, and the left border of flatness may reach or extend beyond the mammillary line, the line of relative dulness extending three or four centimetres farther outside, and the apex beat perhaps appearing in the fourth intercostal space, the upper and lower borders being lifted up.

In inflammatory exudations which have reached a certain height the percussion remains the same over the heart in the dorsal and in the upright position. Hydro-thorax, on the other hand, influences the percussion of the heart only when the patient sits up, provided the fluid is not present in large quantity, because, being freely movable, it gravitates with the patient on the back into the back part of the pleura. When the fluid is in considerable amount, we then find it impossible to define the lateral heart boundaries. Displacement of the heart is exceptionally rare in hydro-thorax—first because the latter is usually double, and second because such large collections of fluid as in pleurisy are extremely uncommon.

(*D*) In simple pneumo-thorax of the left side and in pyopneumo-thorax with moderate exudation the absolute flatness and relative dulness completely disappear on the left of the sternum in the dorsal decubitus, and give place to a tympanitic sound. If either of these conditions occurs on the right side, the tympanitic sound sometimes goes beyond the left edge of the sternum (Weil says that the sound in both these cases is *clear* and not tympanitic), and the absolute flatness appears to be diminished in breadth, while the relative dulness cannot be found on the right of the sternum. Simple pneumo-thorax, with the air under no great tension, does not cause displacement of the heart. If, on the other hand, the tension is great, displacement quickly follows, and is perhaps more considerable than in pleuritic effusion. The signs of displacement are the same in the two cases, but in pneumo-thorax the absolute cardiac flatness can frequently be mapped out against the tympanitic sound.

In the rare cases where gas is present in the pericardium (pneumo-pericardium or pneumo-pericarditis) a tympanitic resonance replaces the flat and dull cardiac sound in the dorsal decubitus. When the patient sits up or leans forward the resonance over the lower portion is dull, because the heart and any fluid present sink forward and downward (Weil).

In emphysema of the anterior mediastinum there is a tympanitic resonance in the region of normal cardiac flatness, but it is less loud than in pneumo-pericardium. The borders of relative dulness in such cases may remain normal.

In dexiocardia the absolute flatness is absent on the left, but is found on the right in exactly reversed position. The same is true of the relative dulness.

(*E*) Enlargement of the entire cardiac dulness in all directions, or chiefly on one or the other side, comes from enlargement of the whole heart or of portions of it, or from the collection of fluid in the pericardium. It must be borne in mind that hypertrophy alone cannot cause

such an increase in size as to be absolutely proved by percussion. A marked increase of the relative heart's dulness points rather to dilatation of the portion of heart affected or of the whole organ, and there is with this usually an hypertrophy of the muscle. So that we speak of this condition as *excentric hypertrophy*, in which sometimes hypertrophy, sometimes dilatation, preponderates.

In excentric hypertrophy of the right ventricle it is dilatation of the right auricle chiefly which gives rise to the increase of cardiac dulness to the right. The left auricle is not directly accessible to percussion, but a dilatation of it may contribute to an increase of the cardiac absolute flatness by pushing the whole heart somewhat more forward and the apex to the left and downward.

In hypertrophy and dilatation of the right ventricle alone, with its accompanying dilatation and hypertrophy of the right auricle, the apex beat being in essentially normal position, we find the right border of relative dulness farther than normal from the middle line in the adult—more than four and a half centimetres; that is, about in the right parasternal line or near it. If the hypertrophy and dilatation are extreme, the right border of relative dulness may be between the parasternal and mammillary lines, reaching the latter only in rare cases. The boundaries are normal on the left of the sternum. The left and lower borders of absolute flatness also remain normal, but the right border in great degrees of excentric hypertrophy runs obliquely from the inner end of the upper border down to the middle or right edge of the base of the xiphoid cartilage; but in lesser degrees it may retain its normal place at the left edge of the sternum.

In hypertrophy and dilatation of the left ventricle alone one of the most important signs is displacement of the apex beat outward and downward. Corresponding to this, the left border of relative dulness lies in the fifth intercostal space and on the sixth rib three or four centimetres outside the mammillary line, and may even reach the anterior axillary line. The upper border is frequently higher than normal, at the lower border or middle of the second costal cartilage; the right border is at the normal place. The lower border corresponds to the apex beat, and is deeper than normal, running down in an oblique line. The absolute flatness is increased toward the left. The left border approaches nearer the mammillary line—reaches or goes beyond it not infrequently. The upper border may either be at the normal level or higher, about in the third intercostal space or lower border of the third costal cartilage. The right border is formed by the left sternal edge, as in the normal condition. The height of both the relative and absolute areas is therefore increased, and at the same time the left border extends farther from the middle line. Jürgensen says that predominating dilatation gives a more curved left border than predominating hypertrophy, and that in existing hypertrophy the onset and progress of dilatation can thus be demonstrated.

Combined hypertrophy and dilatation of both ventricles, including the same of the right auricle, may be inferred where there is great displacement of the apex beat downward and outward, with, at the same time, the right border of relative dulness more than five centimetres distant from the median line.

The greatest increase of both relative dulness and absolute flatness is observed in insufficiency of the mitral valve, which has led to hypertrophy of both right and left ventricles, and where from overstrain of a weak and poorly nourished heart simple dilatation of both ventricles has followed. In such cases, however, the apex beat is not displaced so far downward as in extreme hypertrophy of the left ventricle alone.

Fluid in the pericardium, either the result of inflammation (pericarditis) or a symptom of general dropsy (hydropericardium), often leads to very considerable increase of the relative cardiac dulness and absolute flatness. The degree of the same of course depends on the amount of fluid. The enlargement takes place, as a rule, in all directions, but is greatest to the right and left. Rotch from his experiments regards flatness in the right fifth intercostal space five centimeters from the right border of the sternum as diagnostic of fluid in the pericardium.

In very great effusion the right border of relative dulness may be in the right mammillary line or even outside it, the upper border at the level of the second pair of costal cartilages or even higher to the first left costal cartilage, the left border several centimetres outside the left mammillary line or even in the anterior axillary line, while the lower border runs from the right mammillary line at the level of the sixth intercostal space to nearly the left anterior axillary line at the level of the seventh rib or even seventh intercostal space. The left half of the diaphragm, and with it the left lobe of the liver, are displaced downward somewhat in this case. The whole upper border from the right mammillary line through the second pair of cartilages or first intercostal space to the left border forms usually a curved line. The border of absolute flatness is parallel to the above, and usually only at a short distance inside it. If the overlying lung has become airless from pressure, the relative dulness of course disappears and we only get the flat sound.

If the collection of fluid is less considerable, the percussion areas are essentially the same as in dilatation of both ventricles. A point of diagnostic value in pericardial exudation or dropsy is that in these conditions the absolute flatness in the upright position is, as a rule, greater, often markedly so, than in the dorsal decubitus, because in the latter the heart, together with the surrounding fluid, gravitates and allows the lungs to distend, while in the upright position the heart, approaching the chest wall, forces the lungs to retract. For the same reason the apex beat in such cases may be apparent in the upright position, and be absent when the patient lies down. With great collection of fluid the apex beat can neither be seen nor felt in any position of the body. When it is demonstrable an important sign of pericardial exudation, according to Gerhardt, is that the left border of absolute flatness and also of relative dulness lies not inconsiderably beyond the point of the apex beat, because the fluid in the pericardium fills out the space beyond the apex.

The shape of the flatness in pericardial exudation is described by most authors as being usually three-cornered, but that given by Eichhorst is trapezoidal, the right border being steep and the left sinking gradually.

If the lung in front of the heart is attached to the chest wall, the

area of cardiac flatness may remain small, even when the pericardium is distended with fluid, or if the lung is still able to retract a little, the flat area may be of irregular shape. In extreme emphysema a considerable accumulation of fluid in the pericardium may give but slight increase of the absolute flatness; the relative dulness, however, will enable us to determine the size of the distention.

(*F*) Dilatation of the aorta from arterio-sclerosis or aneurysm may give rise to a dulness the extent of which depends on the size of the aneurysm. In beginning dilatation of the ascending aorta a small dull area first appears near the right edge of the sternum in the second intercostal space and on the second rib. As it increases and reaches the arch there is absolute flatness in the upper part of the sternum, which may include the entire manubrium.

Auscultatory percussion has been advised for determining the cardiac boundaries, but it does not seem to present any advantages over the ordinary methods, and is not, therefore, recommended to the general practitioner.

Auscultation.—In the examination of the heart and bloodvessels it is best to make use of the stethoscope, and of the different forms of that instrument the binaural one devised by Camman is the most satisfactory.

1. *Normal Condition.*—When we listen over the heart we hear everywhere during the time of a single heart movement two sounds, known as the first and second cardiac sounds. Under pathological conditions we hear, instead of or in addition to the normal sounds, others called murmurs, either at isolated places or over the whole cardiac area.

If the stethoscope is placed over the apex beat, two sounds are heard rapidly following each other, separated by a very short pause. The first, or systolic, synchronous with the impulse, is stronger and longer; the second, or diastolic, is feebler and shorter, so that the rhythm is that of a trochee. A longer pause succeeds the second sound. Then the systolic sound occurs again, succeeded by the short pause and the second sound and long pause in regular order. If the stethoscope is placed over the lower end of the sternum or at its right edge in the sixth intercostal space, the heart sounds are heard in the same rhythm. The sound heard here springs from the right, that at the apex from the left, ventricle.

Auscultation at the left on the third costal cartilage or in the second intercostal space close to the sternum gives two sounds: the first, synchronous with the apex beat, is feebler, the second stronger and more accentuated. The rhythm is that of the iambus, but with the accent on the second sound, and the first sound longer than the second. The two sounds are also heard on the right in the second intercostal space close to the sternum. In these two places, the first corresponding to the pulmonary opening and the second to the aortic opening, the sounds and pauses maintain the same length as at the apex and the lower end of the sternum.

The first sound corresponds in time to the ventricular systole and has a double origin: (1) the change of tension which the auriculo-ventricular valves undergo when they close the mitral and tricuspid orifices, and (2) the muscle sound caused by the contraction of the

ventricles. It is still a question whether the impaction of the heart against the chest wall enters into the production of the first sound. The first sound is low in pitch, long in duration, and is described as booming in quality. It is heard all over the heart, as mentioned above, and often even beyond its limits, but is loudest at the apex, where it is attributable to the mitral valve. The first sound heard over the middle of the lower part of the sternum is attributable to the tricuspid valve.

The second sound follows the first, as has been stated, almost immediately, and is attributable to the vibrations set up in the aortic and pulmonic valves by the change of tension to which they are subjected at the time of closure. Compared with the first sound, it is short, sharp, and valvular. It is heard all over and beyond the cardiac area, but is loudest at the places mentioned above—namely, the second left intercostal space or third rib, and the second right intercostal space, both close to the sternum. The sound on the right comes from the aortic valves, that on the left from the pulmonic valves.

When all the valves and cardiac muscles, including the papillary muscles, are normal, the heart sounds in all persons and ages maintain the same rhythm; the strength and loudness of the sounds varies, however, with the individual under different conditions.

The sounds are louder in excited than in quiet cardiac action, as may be demonstrated by causing the patient to move about vigorously after sitting still. They are louder in the upright than in the reclining position. They are decidedly less loud after prolonged rest in bed. The thickness of the thoracic walls has some influence on the loudness of the sounds, the latter being more marked in thin than in fat people, and a largely developed female breast causes marked diminution of the intensity.

2. *Pathological Changes in the Cardiac Sounds.*—The following deviations from the normal are observed in the cardiac sounds: they may be either strengthened or weakened; there may be disturbance of the rhythm or a reduplication of the sounds.

A strengthening or a weakening of both sounds at the same time may be met with either at all the orifices in the same degree or it may predominate at any one of them.

Strengthening of all the sounds is found in pathological increase of the cardiac activity in nervous palpitation, Basedow's disease, and in febrile conditions which have not caused cardiac weakness. The sounds also appear loud when the heart lies against the thoracic wall over a greater area than common, as in deformities of the thorax which lead to flattening of the front left chest, or in uncovered heart from retracted lungs. The pulmonic second sound in the latter case is often louder than the aortic second.

A general weakening of the cardiac sounds is observed in all enfeebled conditions of the heart, as after great hemorrhage, protracted febrile diseases, in anæmic or otherwise enfeebled patients, in exhaustion of the heart after over-exertion, in simple dilatation, degeneration of the cardiac muscle, especially fatty heart, and chronic myocarditis. In all such cases the cardiac sounds are weak, corresponding to the disturbance of nutrition or degeneration.

During a fainting fit and in collapse after great loss of blood the

sounds become so faint sometimes that strict attention is necessary to hear them. At the same time, the diastolic pause is somewhat prolonged and the rhythm thereby altered. A similar condition is found in many cases of cardiac paralysis after diphtheria. An enfeeblement of the sounds is also observed in sudden or gradual collapse in the course of febrile diseases, accompanied usually by a quickening of the heart beats and shortening of the pause. In the collapse of cholera the cardiac sounds get weaker, till at last the second, and finally the first, sound becomes inaudible.

Great Disturbances of Rhythm.—Arrhythmia frequently follows degeneration of the cardiac muscle, especially chronic myocarditis and sclerosis of the coronary arteries; also certain conditions of poisoning, as from digitalis or nicotine; and in the last stages of severe infectious diseases. In such cases we find double sounds following each other, sometimes very rapidly, sometimes very slowly, with the greatest irregularity, and accompanied by abnormally short or long pauses. The length of the pause may even at times exceed the duration of the whole heart beat. Following this, for a time the sounds may be so rapid that we can scarcely distinguish a pause between them, and in the next instant there may be a succession of normal sounds and pauses. When the heart beats follow each other rapidly we frequently hear at the apex only a single sound, the systolic, and at the same time on feeling the pulse find that some beats are missed at the wrist, though we hear the sound at the apex. In other cases instead of the double sound we may find three short sounds rapidly following each other—the gallop rhythm, with the accent either on the middle or last sound. This may continue for some minutes. Lesser degrees of arrhythmia are observed where there is only an occasional dropping of a beat, the sounds following each other regularly at other times.

Great feebleness of the heart sounds can only be attributed to a weak heart when there is nothing which prevents the conduction of sound to the ear. If such conduction of sound is interrupted, even a powerful heart appears weak. That which is oftenest the cause of poor sound transmission is the heart extensively covered by lungs, so that the orifices and apex are separated from the thoracic wall by a thick layer of lung. In great pulmonary emphysema the cardiac sounds of the arterial orifices and apex are, as a rule, feeble, and if there is a weak heart at the same time, the sounds can scarcely be heard. The only place where they can be distinctly heard in extreme emphysema is the lower end of the sternum or xiphoid cartilage.

A less frequent cause of weakening of the heart sounds is the occurrence of fluid between the heart and chest wall, as in pericardial exudation and hydro-pericardium, and in great pleural effusion of the left side.

In large pericardial exudations the sounds often can scarcely be heard at the apex and lower end of the sternum, and at the arterial orifices they are feeble, though more distinct in the upright position, and especially when the patient bends forward, than when on the back. In great exudations which have come on rapidly, but have not yet led to extreme heart weakness, the very low sounds of the heart are in striking contrast to the strong pulse, and thus furnish a point of value for the diagnosis in doubtful cases between pericarditis and dilatation of the heart.

In very large pleural exudations on the left side, where the heart is pushed to the right and separated from the left chest wall, the cardiac sounds sometimes are inaudible to the left of the sternum, but they are heard very loud to the right of the median line. Right-sided exudations only cause the sounds to disappear from the right of the sternum, corresponding to the displacement of the heart.

In those diseases, on the other hand, which offer favorable conditions for their conduction the cardiac sounds are often increased, and can sometimes be heard at a great distance from the heart and in places where they are not otherwise perceived. Among such pathological conditions are compression of the edges of lung covering the heart, dependent on the pushing up of the diaphragm by ascites or large abdominal tumors, where the sounds are very loud at the apex and the arterial orifices. Phthisis of the left upper lobe causes the sounds in the vicinity of the pulmonary orifice to be increased and enables them to be heard to a great distance, perhaps even as far as the first intercostal space. The same thing is observed in pneumonia of the left upper lobe, but less frequently in so striking a manner. In pneumonia or phthisical infiltration of the right upper lobe such an increase and transmission of the cardiac sounds is much more infrequent. In pneumonia or hypostasis of the lower lobes it is by no means rare to hear the cardiac sounds distinctly both on the right and on the left behind near the vertebræ.

Strengthening of Single Sounds.—In some cases we hear only a single sound increased at the ordinary places for auscultation, the others being normal or sometimes even weakened. Such a sound is said to be accentuated. This is the case when the closure and tension of the valves at one orifice is caused by an increased pressure of the blood. For example, we find with a normal condition of the valve the first sound at the apex strengthened in hypertrophy of the left ventricle, the first sound increased at the lower end of the sternum in hypertrophy of the right ventricle, the second aortic sound strengthened in increase of the resistance in the arterial system of the greater circulation, the pulmonary second sound strengthened in increased resistance in the pulmonary artery or lesser circulation.

The purest form of strengthening of the first sound is found in hypertrophy of the left ventricle accompanying granular atrophy of the kidneys and the arterio-sclerosis of the aorta without valvular lesions. In the hypertrophy which follows stenosis of the aortic orifice, besides the strengthened first sound at the apex, there is heard a faint transmitted systolic murmur.

The purest strengthening of the first sound over the right ventricle is heard in cases of pulmonary emphysema and pulmonary contraction, while the strengthened first sound in hypertrophy of the right ventricle from insufficiency of the mitral valve is frequently concealed, or at least rendered indistinct, by a transmitted murmur from the mitral orifice.

The second aortic sound compared with the second pulmonic sound appears accentuated in granular atrophy of the kidneys and in arterio-sclerosis of the aorta if the latter has not led to insufficiency of the aortic valves and the appearance of a diastolic murmur. The accen-

tuated second aortic sound often has a metallic ring in atheromatous degeneration.

The second pulmonic is stronger than normal, and louder than the aortic second in emphysema and pulmonary shrinkage. It is also accentuated in all diseases of the heart and its valves which lead to hindrance of the flow of blood from the pulmonary veins into the left auricle, causing passive congestion of the pulmonary veins and capillaries and offering resistance to the blood current in the pulmonary arteries. For this reason accentuation of the pulmonic second sound is a necessary sequence of valvular disease at the mitral orifice, whether it be insufficiency or stenosis. It may also occur in all other disturbances of the heart's action resulting in stasis of blood in the small circulation, as in degeneration of the cardiac muscle, aortic valvular disease, and contracted kidney, where compensation is lost through degeneration of the hypertrophied left ventricle.

While accentuation of the pulmonic second sound is an aid to diagnosis in emphysema and pulmonary shrinkage, it is nevertheless in most cases only an incidental occurrence. In the diagnosis of valvular disease of the heart, on the other hand, it is often of the greatest significance, and forms a sign of far greater diagnostic value than the enlargement of the relative dulness in one or the other direction obtained by percussion. As we shall soon see, a systolic murmur at the apex may occur when the mitral valve is not diseased, and therefore not permanently incompetent, and with it there may not infrequently be found an increase of the cardiac dulness, especially to the right, due to degeneration or exhaustion of the heart muscle. But if, besides a systolic murmur at the apex, accentuation of the pulmonic second sound is found which cannot be explained by retraction of lung, it is proof that there is increased resistance in the lesser circulation, and we may conclude with great probability that the cause is a hindrance to the flow of blood from the pulmonary vein into the left auricle, and that there is a permanent disturbance of the mitral valve—namely, incompetence.

Division or reduplication of heart sounds is not rarely caused by slight disturbances of the heart's action, though frequently serious pathological changes of the cardiac muscle or valves lie at the bottom of it.

It is called division when in place of one sound two sounds occur, separated by a pause of minimum duration; it is called reduplication when the pause lasts longer. In the latter case the duration may almost equal the normal pause between the two heart sounds, so that reduplication of the first sound at the apex gives the impression as if three short sounds followed each other in almost equal times, the accent being either on the middle or last sound—the gallop rhythm.

Division or reduplication affects the first sound most frequently, more infrequently the second sound. With normal valves we find the first sound in the one case, and the second sound in the other, sometimes both, divided, most frequently in excited cardiac activity and in nervous, irritable people. The former, as a rule, is distinct only at the apex and lower end of the sternum, the latter only at the arterial orifices, most distinctly at the pulmonary orifice. A well marked gallop rhythm may sometimes be observed under the same conditions in otherwise healthy people with anatomically intact hearts. More frequently, however, it

occurs in great cardiac weakness, in severe disturbances of compensation in the terminal stages of valvular disease, and in diphtheritic cardiac paralysis; and in such severe conditions of disease is always to be regarded as a grave sign.

The cause of the division or reduplication is undoubtedly in the one case the fact that the two auriculo-ventricular valves, and in the other the pulmonary and aortic valves, do not experience their highest degree of tension exactly at the same time. According to Potain, one hears the first half of the divided first sound louder at the apex, the second half loudest over the lower end of the sternum, and of the divided second sound the first half louder at the aorta, and the second over the pulmonary artery. We must therefore conclude that in such a case the tension of the tricuspid follows somewhat later than that of the mitral, and the closure of the pulmonary valves is later than that of the aortic valves.

A metallic ring or timbre of the first sound at the apex is sometimes heard in increased cardiac activity when the heart is normal, but it is more common in hypertrophy of the left ventricle. Metallic ring of the aortic second sound indicates, as a rule, atheromatous degeneration of the arterial wall. A stomach full of gas may give a metallic ring to the two sounds at the apex, as may also a pneumo-pericardium, a pneumo-thorax, or even a large smooth-walled cavity of the lung lying close to the heart.

3. *Cardiac Murmurs.*—We distinguish endocardial and exocardial or pericardial murmurs. The former arise inside the heart, the latter outside it, usually between the two layers of pericardium.

Endocardial Murmurs.—Endocardial murmurs may be caused by—(*a*) true organic or anatomical changes of the valves, and are therefore called organic murmurs; or (*b*) they may appear in connection with nutritive disturbances of the heart, primarily through disease of the cardiac muscles or vessels, or secondarily accompanying anomalies of the blood or severe general diseases, and are called inorganic murmurs.

(*a*) *Organic Murmurs.*—Pathological changes in the valves give rise to murmurs when conditions are thereby produced in which the blood flows with sufficient rapidity through a narrow opening into a wide space. If the conditions are such that this flow of blood occurs during the systole of a ventricle, and is caused by it, there is a systolic murmur. If, on the other hand, it is caused by the ventricular diastole, there is a diastolic murmur. If the systole of an auricle (almost always the left) furnishes the current, a presystolic murmur is produced.

A systolic murmur occurs in the ventricles, or, to speak more exactly, in the auricles, if the auriculo-ventricular valves are incompetent, thereby allowing in systole of the ventricles a part of their contents to be thrown back into the auricle through the open fissure of the valve, the fissure in the valve, however wide it may be, always presenting a narrowing in relation to the auricle.

A diastolic or presystolic murmur occurs in the ventricles if the auriculo-ventricular ostium is narrowed, the blood flowing through the narrow ostium into the wider ventricle during ventricular diastole. The murmur is diastolic when the ostium is so narrow that the slow current with which the blood flows into the ventricle during diastole is never-

theless sufficient to produce a murmur owing to the great difference between the ostium and ventricle. It is presystolic when with a less marked narrowing of the ostium the increased rapidity of the diastolic current due to auricular contraction, and coming in the presystolic period, is sufficient to produce a murmur.

At the arterial orifices a systolic murmur can occur only with stenosis of the same, the stream of blood being driven forward by the systole of the ventricle through the narrow opening of the orifice into the wider lumen of the artery. A diastolic murmur can occur at this point only when the semilunar valves are incompetent, the forward pressure into the arteries ceasing with the beginning of the ventricular diastole, and the resistance in the arteries and capillaries causing a backward flow through the narrow fissure of the valves into the ventricle and causing the diastolic murmur.

Insufficiency of the valves and stenosis of the ostia are usually caused by the same pathological processes—namely, chronic endocarditis at the auriculo-ventricular valves, at the aortic valves partly chronic endocarditis, partly endarteritis (the latter sometimes being syphilitic), at the pulmonary valves partly endocarditis, partly congenital malformations. The insufficiency of the valves may be merely relative, from dilatation of a ventricle or artery, the points of insertion of the valves being so far removed from each other that the lines of closure no longer reach each other, but leave a fissure between them, through which the blood flows back into the auricle in systole or into the ventricle in diastole. Relative insufficiency is met with most frequently at the tricuspid valve following protracted difficulty of emptying the right ventricle, which has led to great dilatation of the same.

Endocarditis in extra-uterine life affects the left side of the heart almost exclusively, and endarteritis of marked degree is far rarer in the pulmonary artery than in the aorta. Acquired valvular lesions, therefore, almost always occur in the mitral or aortic valves, and, with the exception of relative insufficiency, valvular lesions or stenosis at the pulmonary orifice or tricuspid are, as a rule, congenital.

In chronic valvular lesions insufficiency of the auriculo-ventricular valves, as a rule, is caused by shrinking or shortening of the extremity of the curtain, and frequently also by shortening of the chordæ tendineæ. It is easily seen that such a shortening of the valve segment alone may be sufficient to prevent the closure of the ostium during systole. This is still more evident when the chordæ tendineæ have likewise become shorter than normal through shrinking and thickening, thus preventing the auricular surfaces of the valves from approaching each other.

Much less frequently a great loss of substance at the edge of a valve or a perforation of the same from ulceration is the cause of insufficiency. Still rarer are the destruction and separation of one or more tendons from the same process, which allows the valvular segment to flutter and fold back into the auricle during systole of the ventricle.

A moderate stenosis of the auriculo-ventricular orifice may arise as follows: The valvular segments, having become contracted and stiff, no longer hug the wall during ventricular diastole, as they do in their normal delicate condition, but extend into the lumen as stiff projections,

leaving a fissure between them which does not open much wider than during systole. The stenosis is more marked when the edges of the valves are adherent to each other near their insertion in greater or less extent. It is not rare to find a stenosis of the ostium combined with insufficiency of the valves.

Insufficiency of the aortic valves also frequently arises from shrinking of the same, and if endarteritis is the cause, there is often ulceration as well, leading to loss of substance at the edges of the valves, or in rare cases to perforation of one or other of the segments. The valves may become adherent to each other at the periphery of the vessel, and also give rise to stenosis of the orifice.

The pulmonary valves may become insufficient from similar changes when a fœtal endocarditis has caused them to be diseased. The same is true for the rare cases in which they are attacked by endocarditis at any time of life after birth. Congenital insufficiency of the pulmonary valves is more frequent (though still rarely observed), due to rudimentary development of one or more of the segments during fœtal life. Stenosis of the pulmonary ostium, the most frequent of congenital heart lesions, may be caused by changes in the valves; as, for example, adhesions following fœtal endocarditis or congenital narrowness of the artery, particularly of the conus arteriosus.

Insufficiency of the tricuspid valve and stenosis of its orifice, if endocarditis was the cause, comes from the same changes in the valves and chordæ tendineæ as we have already seen in the mitral valve. But this valve is almost as rarely attacked by endocarditis in fœtal as in extra-uterine life. Relative insufficiency of the tricuspid has already been described.

As already stated, in insufficiency of an auriculo-ventricular valve, most frequently the mitral, there is a systolic murmur which comes from the eddying current which is produced by the regurgitant stream of blood in the ventricular systole going through the more or less narrow fissure of the valves into the auricle.

In stenosis of the mitral ostium the blood stream which produces the murmur has the opposite direction. The eddying movements occur during diastole or presystole below the valve fissure in the cavity of the ventricle.

The vibrations of the fluid give rise to vibrations or thrills in the wall of the ventricle, which can frequently be felt at the apex as frémissement cataire. It is evident that the thrills produced by diastolic murmurs are much oftener distinctly felt at the heart's apex than those produced by systolic murmur, since the former are directly transmitted to the wall of the ventricle, while the latter affect the wall of the auricle, from which it must be transmitted by the ventricular wall to the apex.

The regurgitant blood stream producing the diastolic murmur in insufficiency of the aortic valves gives an eddying current below the semilunar valves, chiefly in the conus arteriosus; and it is therefore over this that, as a rule, the murmur is heard most distinctly. The same thing is true of insufficiency of the pulmonary valves.

In stenosis of the aorta or pulmonary ostium, on the other hand, the eddy arises above the semilunar valves in the first part of the

artery in the blood driven through the narrow orifice during the ventricular systole.

Under favorable conditions the vibrations of the wall of the vessel can be felt through the thoracic wall in these valvular lesions also. This is especially the case in pulmonary stenosis, in which not rarely, when the heart is somewhat uncovered by lung, a systolic thrill may be distinctly felt in the second or third left intercostal space close to the sternum. Less frequently the thrill may be felt in stenosis of the aortic ostium to the right of the sternum in the second intercostal space. This probably only occurs when the aorta is dilated above the stenosed point.

In combined valvular lesions the murmur may occur either in one and the same ostium both in systole and diastole, or at different ostia at the same time in systole or diastole, or at one ostium in systole and in the other at diastole. Frequently there is combined insufficiency and stenosis of the mitral. Also in insufficiency of the aortic valves there exists not infrequently at the same time stenosis. In the first case there are at the apex and in the second at the aortic orifice two murmurs to be heard, a systolic and a diastolic, of about equal intensity. Besides insufficiency of the aortic valves there may also be insufficiency of the mitral, and with a stenosis of the mitral orifice an insufficiency of the tricuspid (most frequently relative), and accordingly with the diastolic murmur at the aorta there may be a systolic murmur at the apex or base of the left ventricle, or with a diastolic murmur at the apex there may be heard a systolic one over the right ventricle.

To an insufficiency of the mitral valve or to a stenosis of the pulmonary ostium there may come as the result of a great dilatation of the right ventricle a relative insufficiency of the tricuspid, and with a stenosis of the aortic ostium there may be an insufficiency of the mitral. Accordingly, a systolic murmur may be heard at the same time at the pulmonary ostium and at the right ventricle, at the right ventricle and apex, and at the aortic orifice and the left ventricle. Stenosis of the pulmonary valves combined with insufficiency of its curtains is more uncommon; in such cases a systolic and a diastolic murmur occur together at this ostium.

But in all valvular lesions, no matter of what kind, a murmur can only occur when the chief conditions are fulfilled—namely, when the blood is driven with sufficient force and rapidity through a narrow opening into a wide space; and where this is not the case even with existing valvular lesions no murmur is produced. Therefore in such cases in great cardiac weakness we sometimes find no murmur where one was distinctly heard before. This is occasionally observed in fatigue of the heart after great exertion and in disturbances of compensation, especially toward the end of life or during the death agony.

Great rapidity and irregularity of the heart's action have a similar influence in preventing the production of a murmur through insufficient diastolic filling of the ventricle, so that too little blood is passed through the diseased ostium in the unit of time, whether it be in systole or diastole. In great irregularity of the cardiac movements one often hears at one and the same place with some heart beats a distinct murmur, with others a sound. Murmurs may also appear in great rapidity of the

heart, so that it is difficult to determine the time of the murmur, and the slowing action of digitalis on the heart has to be awaited to determine whether it is systolic or diastolic.

Sometimes in a heart that is still powerful a murmur becomes inaudible while the patient sits or lies quietly, because of the slight energy of the cardiac movements; if, however, the movements are made energetic by muscular exertion or mental excitement, it comes out distinctly.

The murmur occurring in acute endocarditis does not admit of so easy an explanation as that in a marked valvular lesion, for it occurs so soon after the disease sets in that there can be no possibility of the shortening of the tip of a valve segment or of a chorda tendinea, the common causes of murmurs in chronic valvular lesions. From the beginning of the endocarditis to the occurrence of a distinct mitral systolic murmur, the mitral valve being the one almost always involved, is only a few days. In this time, as has been repeatedly observed, there are only papillomatous vegetations or verrucose growths found on the auricular surface of the mitral valve, especially in the line of closure of the valve. According to the belief of the present day, these vegetations cause either an insufficiency of the valve or a stenosis of the ostium, just as in the chronic forms. That such a rapidly occurring disturbance of function is really the cause of the murmur is proved by the fact that distinct accentuation of the pulmonic second sound follows often in a few days. The usual explanation offered of the murmur is that the vegetations occurring at the lines of contact of the valve disturb the nice adjustment of the same, and allow of small holes through which there is regurgitation during ventricular systole.

In endocarditis of the mitral valve a diastolic murmur, coming on simultaneously with the systole or occurring alone as early significant of stenosis of the ostium, is not common. It is only exceptionally caused by vegetations, which, as a rule, are not large enough to produce a narrowing of the lumen sufficient to cause the murmur. As a rule, the stenosis depends on adhesion or firm sticking together of the valvular edges, which requires more time to bring about.

The rare cases of endocarditis of the tricuspid are explained in the same way as in the mitral. If the aortic valves—or, far more infrequently, the pulmonic valves—are the seat of the disease, vegetations produce stenosis of the ostium more readily. In a rapidly arising insufficiency of the semilunar valves the same process probably occurs as in the similar disturbance of function of the mitral.

Mitral insufficiency in acute endocarditis might perhaps be also caused by the accompanying myocarditis of the papillary muscles interfering with the simultaneous and symmetrical tension of the chordæ tendineæ. Nevertheless, the fact that vegetations are present in every case of valvular endocarditis; further, that a murmur once occurring and accompanied by accentuation of the pulmonary second sound does not disappear even temporarily; and, lastly, the fact that in the gradual and latent-occurring shrinking of the valves the character of the murmur is not changed,—all indicate that the insufficiency as well as the murmur are from the commencement caused by valvular disease.

The murmurs which arise in the course of an acute endocarditis at

any one of the ostia are therefore to be explained just as those following chronic valvular lesions—namely, as due to insufficiency of valves or stenosis of ostia.

It is usually not difficult to determine which phase of the cardiac movements a murmur belongs to; sometimes, again, it is not easy, and there are cases occasionally where it is impossible, on the instant, to definitely decide the question. The latter is more particularly the case when the heart's action is very rapid. Cardiac murmur follows cardiac sound and sound murmur so directly that the rhythm cannot be distinguished, and we are obliged to depend on the effect of rest, the use of ice, digitalis, or strophanthus, to slow up the heart sufficiently to enable us to make the diagnosis.

The length of the duration of the murmur is variable, and, as a murmur does not always fill out the whole time of the systole or diastole, the additional term presystolic has been accepted generally as indicating the last short portion of the diastolic pause following active contraction or systole of the auricles. A presystolic murmur, therefore, may be the only sign of stenosis of the mitral ostium, rarely of the tricuspid ostium.

If the heart movements are not too rapid, it usually is not difficult to determine the time of a murmur at the arterial ostia, most frequently found at the aorta, while in valvular lesions of the auriculo-ventricular ostia, especially at the mitral, this determination is far more frequently attended with difficulty.

The rhythm is more distinctly marked normally at the arterial ostia, and comparison with the other large artery is easy, so that this usually gives instant information as to the time of the murmur. The diastolic murmur is the easiest one to recognize here, because the iambic rhythm either remains distinct or becomes more marked than normal. Also in a systolic murmur the diagnosis is rendered easier by the accentuation of the second sound or by comparison with the neighboring arterial ostium.

Mitral or tricuspid murmurs are much more difficult to time in certain cases. It is always well to compare their time with the carotid pulse or apex beat. A murmur which occurs at the same time with the carotid pulse must be systolic; if it is directly after, it is diastolic. Perhaps an easier way of determining the rhythm is to start from the apex and listen with the stethoscope, step by step, up to the pulmonary orifice; the nearer the latter is approached the more distinct becomes the second sound, and it can be readily determined if the murmur precedes this or is synchronous with it. In a strongly heaving apex beat this may be utilized to help determine the time, the head being lifted thereby.

When two murmurs are heard at the same place, one must be systolic and the other diastolic or presystolic. A diastolic murmur is separated from the following systolic one by a distinct pause; a presystolic murmur immediately precedes the systolic one.

Having determined the time of the murmur, the next thing is to find where it is the loudest, which answers the question at which ostium it occurs and which valve is diseased.

A murmur heard loudest at the apex indicates lesion of the mitral

valve; one heard at the lower end of the sternum or somewhat to the right of this, louder than at the apex, indicates lesion of the tricuspid valve; if it appears loudest at the second intercostal space to the right of the sternum, it is a lesion of the aortic valve; and when it is heard loudest at the second intercostal space or third costal cartilage to the left of and near the sternum, it is lesion of the pulmonary valve.

The following table presents the matter more clearly:

Time of the murmur.	Place where best heard.	Valvular lesion.
Systolic	Apex of heart	Insufficiency of the mitral valve.
Diastolic or presystolic . . .	Apex of heart	Stenosis of the mitral orifice.
Systolic	Lower end of sternum	Insufficiency of the tricuspid valve.
Diastolic . . .	Lower end of sternum	Stenosis of the tricuspid valve.
Systolic	Second right intercostal space .	Stenosis of the aortic orifice.
Diastolic . . .	Second right intercostal space .	Insufficiency of the aortic valves.
Systolic	Second left intercostal space .	Stenosis of the pulmonary orifice.
Diastolic . . .	Second left intercostal space .	Insufficiency of the pulmonary valves.

It may be stated as a general rule that when two murmurs are heard with equal distinctness, which are separated from each other and are situated at different ostia, there is a combined valvular lesion; the above table will help to determine what it is. There are, however, certain exceptions to this rule; as, for example, the aortic diastolic murmur may be heard equally loud at the aortic orifice and at the lower end of the sternum.

The murmurs which are heard most distinctly at the apex of the heart, and which are referred to the mitral valve, are quite apt to be propagated toward the axilla, and may even be heard in the back below the scapula. Sometimes the murmur is heard at the base of the heart in the third intercostal space, on the left near the sternum, or at the third costal cartilage, quite as distinctly if not more loud and distinct than at the apex. Naunyn says this is especially the case if a strongly developed left auricular appendage largely fills up the space between the mitral ostium and the thoracic wall, and thereby causes a certain amount of retraction of the edge of the lung. But a marked retraction of the left lung without enlargement of the auricular appendage may also give rise to the same thing.

In great dilatation and hypertrophy of the left ventricle or of the whole heart it may happen that at the usual place of the apex beat we may hear pure heart sounds or nearly so, while at the real apex where it touches the chest wall, at the anterior axillary line or just inside it, we hear a loud systolic murmur, which may be propagated to the posterior axillary line with almost equal loudness. Such a case is instructive as showing that a dilated and hypertrophied right ventricle may hinder the conduction to the ear of a murmur arising in the left ventricle.

Both the systolic and diastolic murmurs at the arterial ostia are often distinctly propagated to the great vessels and lateral regions of the neck.

Diastolic murmurs may be propagated from the arterial ostia downward along the sternum to its lower end. Since, however, diastolic murmurs at the pulmonary ostium are rare, and at the tricuspid still

more infrequent, we are justified, as a rule, in attributing a loud diastolic murmur heard over the sternum to its lower end as indicative of insufficiency of the aortic valves, provided it does not distinctly appear to be loudest on the left of the sternum over the pulmonary orifice.

(*b*) *Inorganic Murmurs.*—Pathological changes in the cardiac muscle may give rise to endocardial murmurs independent of disease of the valves. An example of such change is found in the nutritive dilatation of the heart sometimes accompanying febrile conditions, the explanation being that the elevated temperature injures the tonicity of the muscle tissue. The change affects chiefly or exclusively the right side of the heart, and forms the condition of so-called cloudy swelling or fatty degeneration of the cardiac muscle. In the infectious diseases, as in typhoid, typhus, cholera, articular rheumatism, pneumonia, smallpox, scarlet fever, erysipelas, diphtheria, and so forth, the same thing may be found. Certain enfeebling causes may give rise to it, as repeated hemorrhage, prolonged disturbance of digestion, chlorosis, protracted diseases. It has also been observed after poisoning by alkalies or mineral acids. Undue muscular exertion has given rise to the same thing. In all these conditions a systolic murmur has been observed, usually at the base of the heart and over the pulmonary ostium, though sometimes a systolic murmur has been heard at the apex. This murmur is usually of short duration, and entirely disappears under appropriate treatment.

An inorganic mitral systolic murmur sometimes replaces the first sound in fat heart and in chronic myocarditis. In chronic endaortitis, the aortic valves being free from changes, a peculiar ringing quality is sometimes observed in the second aortic sound, while the first sound is dull and may be replaced by a systolic murmur.

An inorganic murmur associated with some phase of the respiratory act is sometimes heard. It is usually systolic in time, and disappears when the breath is held, or at least is greatly modified by it. The murmur is usually loudest at the end of the inspiration or expiration.

Lastly, there are some murmurs which do not have any definite connection.

The anæmic murmurs are associated with impoverished conditions of the blood. They are systolic in time, and are heard chiefly at the base of the heart over the pulmonic area, though such are occasionally heard at the apex. Venous hum in the cervical veins frequently is an accompaniment.

In conclusion, inorganic murmurs are almost always systolic in time, they are not persistent, they have no definite line of propagation, they are limited to the cardiac area, and they are unaccompanied by cardiac enlargement. They are either aortic, pulmonic, or mitral murmurs.

Exocardial Murmurs.—Exocardial murmurs are either pleural or pericardial-pleural friction murmurs.

Pleural friction murmurs are of course at once distinguished from cardiac murmurs by the fact that they correspond to the respiratory movements of inspiration and expiration, and do not correspond with the pulsation of the heart.

Pericardial friction and pericardial-pleural friction murmurs do correspond in frequency with the pulsations of the heart, but, as a rule,

their synchronism is much less perfect than in endocardial murmurs. Pericardial murmurs are double; they have a to-and-fro friction sound, and hence have to be differentiated from the double endocardial murmurs of aortic stenosis and incompetence. The rhythm of the murmurs in question is often variable; it is apt to change from day to day, from one hour to another, or even during the examination of the patient. Alterations in the relative position of the two opposed surfaces of the pericardium, such as may be produced by changes in the position of the patient or the application of pressure with the stethoscope over the præcordia frequently modify the character of pericardial murmurs. These murmurs accompany and may obscure the cardiac sounds, but they do not replace them. As a rule, they have a harsh, grating character; they appear superficial; they are usually best heard over the borders of the heart, but have no special point of maximum intensity. They are usually heard over a limited area, and are not propagated in the course of any particular blood current, as are endocardial murmurs. They appear abruptly, and, as a rule, occur in the course of some general affection, as rheumatism. There are usually pain and tenderness on pressure, especially that directed upward from the epigastrium.

The differentiation between pericardial and pericardial-pleural friction is sometimes very difficult. Pericardial-pleural friction, however, is rare; it occurs when the portion of the pleura which is reflected over the pericardium is inflamed, the movements of the heart rubbing the rough, inflamed pleura against the chest wall or visceral pleura, which is likewise usually inflamed. This friction murmur is, as a rule, more affected by the respiratory movements than ordinary pericardial friction. Walshe states that it is usually increased during expiration. It is even more variable than pericardial friction, and may cease during certain pulsations of the heart. Full and rapid inspiration may cause it to disappear.

PERICARDITIS.

By WARREN COLEMAN, M. D.

DEFINITION.—Pericarditis is an inflammation of the pericardium which may be acute or chronic. It may involve the whole or be limited to a portion of the pericardial surface.

ETIOLOGY.—While it is generally believed that all cases of pericarditis are the result of microbic infection, for the purposes of this article it is thought best to consider its etiology under the headings (1) primary, (2) secondary, and (3) consecutive.

(1) *Primary pericarditis* arises in rare instances independently of a constitutional affection or local process elsewhere, as from exposure to cold, and is called *idiopathic*. Such cases, however, are exceedingly rare, and their existence is even denied by competent observers. They have been reported most frequently in children, and, except in the event of death, where an opportunity was presented to examine the bronchial and mediastinal glands for evidence of tuberculosis or inflammation, a reasonable question may be raised as to the primary nature of the pericarditis. In other cases the pericarditis may have followed an obscure articular or abarticular form of rheumatism which escaped observation or was not brought out in the history, and should be classed with the secondary cases.

Primary pericarditis, however, is most commonly met with in connection with blows upon or wounds of the pericardium and its neighborhood, and falls within the province of the surgeon rather than the physician. The rare cases of pericarditis which follow injury to the œsophagus in swallowing foreign bodies, unless the pericardium itself be wounded, must be classed under the third heading, as must also those cases which do not follow immediately upon the infliction of external injury, and which extend rather by contiguity of structure.

(2) *Secondary Pericarditis.*—By far the most frequent cause of secondary pericarditis is acute articular rheumatism, and it is especially likely to occur in the severer types of the disease. The age at which this form of the disease is most commonly met with is from twenty to thirty years. Cases have been recorded where the pericardial preceded the articular manifestations by several days, though they usually follow them. Rheumatic pericarditis is more common in men than in women, probably because men are more subject to rheumatism. It generally occurs with the first attack, and is rarely associated with rheumatism which has become chronic or in which the acute symptoms have subsided.

The exanthemata are often complicated with pericarditis. When associated with diphtheria it is probably caused by streptococcus infection.

As a disease of middle or advanced life pericarditis is seen most frequently in connection with nephritis or gout. It is more often associated with the chronic than the acute form of nephritis, or with the chronic upon which an acute attack has been engrafted. Under these circumstances it is of a sero-fibrinous nature. Pericarditis arising in a gouty individual is probably due to a complicating nephritis.

Septicæmia and pyæmia are frequent causes of pericarditis, as are also certain local processes which occasionally give rise to systemic infection—*e. g.* erysipelas, phlebitis, osteomalacia, and rarely gonorrhœa.

When pericarditis occurs in connection with purpura or scurvy it is of hemorrhagic character. Cancerous pericarditis may result by metastasis from a primary tumor elsewhere in the body, and may be hemorrhagic.

Among the general diseases which are sometimes accompanied by pericarditis may be mentioned typhus, more rarely typhoid fever, smallpox, intermittent and relapsing fevers. Pericarditis is not uncommon in cholera, dysentery, cerebro-spinal meningitis, and delirium tremens.

Though pericarditis is rare before the age of six, cases have been reported in the fœtus resulting from maternal infection, and in the newborn from a sloughing and septic cord stump.

(3) *Consecutive pericarditis*, resulting from extension of inflammation by contiguity of structure, is observed in pleurisy, pleuro-pneumonia, pneumonia, and pulmonary tuberculosis with the formation of superficial cavities in the region of the pericardium. Ulcerations in neighboring structures may lead to the development of pericarditis—*e. g.* in the œsophagus and stomach. Pericarditis is not infrequently caused by hepatic and subphrenic abscesses or by suppuration of the bronchial or mediastinal glands. Inflammations of the endocardium and myocardium are almost certainly accompanied by pericarditis. Necrosis, other than tubercular, of the ribs, sternum, or vertebral column may produce pericardial inflammation. Thoracic aneurysm also may be mentioned as a cause.

Tubercular pericarditis may be part of a general tuberculosis of the serous membranes or may arise by the extension of a tubercular process in some adjacent structure. Hayem and Tissier assert that tubercular pericarditis is most commonly due to extension by contiguity from caries of the sternum or ribs or from an adherent and tubercular pleura. It sometimes follows tuberculosis of the bronchial or, more particularly, the anterior mediastinal, glands, the bacilli being conveyed to the pericardium through the lymphatic channel.

Tubercular pericarditis is oftenest met with in children, though it may occur at any age. The late Dr. Loomis[1] had notes of a case occurring in a man of middle age in whom the diagnosis of tubercular pericarditis was made by exclusion, and whose subsequent history substantiated the diagnosis. This case was explained on the theory that the tuberculosis had origin in the bronchial or mediastinal glands.

Purulent pericarditis may result from the sero-fibrinous form of the disease. This happens much more rarely, however, in the pericardial than in the pleural cavity. Purulent pericarditis is frequently purulent from the commencement of the disease. Parker and Samuel West

[1] Reported by permission of Dr. H. P. Loomis.

believe that purulent pericarditis may be primary—that is, without suppuration elsewhere in the body. Purulent pericarditis may follow injury to the pericardium or its neighborhood. Pericarditis arising in the course of pyæmia is usually purulent in character. Tubercular pericarditis sometimes becomes purulent from superadded infection with pyogenic organisms. Pericarditis resulting from the irruption of pus into the pericardial sac is invariably purulent.

PATHOLOGICAL ANATOMY.—Inflammation of the pericardium begins, as do inflammations of other serous membranes, by an injection of the bloodvessels and temporary dryness of the membrane. The capillaries become distended and the blood current at first stagnates. With increase in the velocity of the blood and in the calibre of the vessels the membrane assumes the reddened appearance characteristic of inflammatory action. The capillaries rupture in places, with the production of ecchymotic spots. At the same time, the liquid part of the blood escapes and the membrane becomes infiltrated and swollen. The nutrition of the endothelium being interfered with, some of the cells die and are desquamated, others proliferate. In consequence of the changes in the vessels and endothelium the membrane loses its pearly lustre. Transudation of the liquid part of the blood continues. Meeting on the free surface of the visceral or parietal pericardium with wandering cells, some of which have disintegrated, coagulation of the fibrin factors takes place. This results in the formation of a thin continuous layer of greater or less extent with irregular thickenings, which present the appearance of little drops of opaque liquid. These enlarge and coalesce, with a consequent increase in the thickness of the membrane, which looks, as Laennec said, like two layers of butter which have been pressed together and separated.

The process may be limited to one or more points of the pericardial surface, visceral or parietal, or be generally diffused. When the inflammation is partial in extent its seat of election is at the base of the heart near the origin of the great vessels. False membranes are more frequently formed on the visceral layer. Inflammation of the pericardium covering the great vessels may extend into their walls, and if limited here should be regarded as an *aortitis*. Thoracic aneurysms not uncommonly have their commencement in this condition.

The outpouring of lymph continues, and further coagulation may lead to the formation of successive superimposed layers of fibrin, with an additional increase in the thickness of the membrane. The coagulation is generally irregular in extent, and the membrane assumes various aspects. It may be honeycombed or felted or have villous projections. The projections, however, are due to the action of the heart pressing upon and rolling out smaller irregularities of surface. The fibrinous exudate may be relatively "dry," and justify the name "plastic pericarditis," but it never occurs without a liquid effusion, however small in amount. A purely serous effusion rarely occurs with pericardial inflammation. The quantity of liquid effused varies greatly. There may be only a few ounces or it may accumulate to the extent of several pints. The color of the liquid may be light yellow, greenish, brown, or hemorrhagic. The presence of blood may result from an actual hemorrhage into the pericardial cavity through rupture of a small vessel.

Examined microscopically, the effusion is found to contain full-sized desquamated endothelial cells, or smaller and more rounded ones from proliferation; pus cells in variable numbers, in ordinary cases not sufficiently numerous to give rise to any opacity; shreds of fibrin which have been torn off by the movements of the heart; and molecular matter which probably comes from the disintegration of pus cells and fibrin filaments. A moderate number of red blood cells is invariably present, but, as a rule, they do not impart any color to the liquid.

If recovery takes place, there is resorption of the liquid effusion.

The pus cells and other cells, except in purulent cases, undergo disintegration and pass off through the lymphatics. A large amount of the fibrinous exudation may disappear in the same manner. But pericardial inflammation usually leaves traces behind it upon the visceral or parietal layers of the pericardium. It is generally agreed that the *milk patches* so frequently observed at the autopsy table are the result of localized inflammations which may or may not have been recognized during life.

Organization of a part or the whole of the false membrane is of constant occurrence in pericarditis. In the former case patches of newly formed connective tissue will be found on the surface of the heart, over which the endothelium may have grown; there may be thickenings of the parietal layer of the pericardium without adhesions or there may be circumscribed adhesions between the two layers. Again, threads or bands of newly formed tissue may pass from the visceral to the parietal layer. In some instances, where the inflammation has been general, organization extends until the pericardial cavity becomes entirely obliterated.

In *purulent* pericarditis the two layers of the pericardium are thickened and have the appearance of a granulating surface. The pus accumulates in quantities varying from a few ounces to several pints, and is rarely absorbed. It may be discharged externally through a fistulous opening or may break through into the mediastinum or one of the pleural cavities. Occasionally the liquid part of the pus is absorbed and caseation results. The cheesy matter may form the seat of calcareous deposit.

Unless a *tubercular* pericarditis is seen early it is often impossible to distinguish it macroscopically from the sero-fibrinous variety. Along with the formation and growth of the tubercles sero-fibrinous characters may be developed, and, unless caseation forms a prominent feature, the tubercles are not recognized without microscopical examination. In cases where the pericarditis is only a part of a general tuberculosis of the serous membranes death usually comes early enough for a diagnosis to be made. Tubercular pericarditis is of the hemorrhagic type in rare instances. When pericarditis is not associated with any of the causes which ordinarily give rise to it, careful search should be made in the bronchial and mediastinal glands or elsewhere for a tubercular focus. Perhaps some of the idiopathic cases are really of this nature. Typical cases of tubercular pericarditis are found, however, with tubercles and caseation, though a certain amount of ordinary inflammatory action is associated with them.

In rare cases inflammation within the pericardial cavity is compli-

cated by inflammation of the mediastinal connective tissue. A tubercular pleura is usually the cause of the inflammation in both situations. These cases, called mediastino-pericarditis, often end in adhesions of the pericardium externally.

As a result of the changes on the surface of the heart its muscular fibres undergo a greater or less amount of parenchymatous degeneration or an interstitial myocarditis may be excited. Myocardial changes most frequently occur in hemorrhagic and purulent pericarditis. If the pressure upon or constriction of the coronary arteries has been sufficiently great, the heart muscle will suffer more generally and dilatation may result. Some authorities state that the heart is always left in a weakened condition after pericarditis. Dilatation with subsequent hypertrophy is frequently met with after extensive adhesions between the two layers of the pericardium.

Symptoms.—The frequent association of pericarditis with other diseases whose symptoms vary in their nature and severity oftentimes renders it impossible to make a diagnosis from the symptoms alone. On the other hand, the symptoms of pericarditis are often so slight that they may be easily overlooked. This is dependent to a certain extent upon the varying intensity of the inflammatory action, and whether it is limited to a portion or affects the whole of the pericardial surface.

In idiopathic cases and in those which are the precursors of acute rheumatism the onset of pericarditis may be marked by rigors or a distinct chill, accompanied by a rise in temperature. There are malaise, anorexia, and perhaps headache and dizziness. The heart action becomes excited and there is a tendency to palpitation. The pulse is full at first, and its frequency is increased. The respiration is hurried in accordance with the change in the pulse rate. On the other hand, there may be no symptoms whatever to indicate the setting in of pericardial inflammation. Its development is insidious, or attention may be first drawn to the heart by a sense of uneasiness in the præcordial region or of constriction around the chest.

The thermometric record is of little value in establishing a diagnosis of pericarditis. It is influenced more by the disease with which the pericarditis occurs than by the pericarditis itself. Charcot observed that the inception of pericarditis in the aged is often indicated by a distinct subnormal lowering of the body temperature, and the same fact has been noted by Lorain in acute articular rheumatism. Hyperpyrexia is most likely to occur in association with acute articular rheumatism.

In other cases pain of a dull, aching, or sharp, lancinating character may be the first symptom manifested. It is often comparable to the pain of pleurisy, but is referred to the præcordium. Sometimes the pain shoots to the shoulder and down the left arm. The pain, however, is by no means of constant occurrence. Associated or not with the pain, there may be præcordial or epigastric tenderness. Pressure on the ensiform cartilage especially is likely to cause suffering or increase it. The pain is sometimes increased by inspiration. This is due to the movement of the diaphragm, and leads to shallow, hurried respiratory acts of the superior costal type. As the liquid effusion accumulates the severity of the pain diminishes. The heart action becomes turbulent, and the pulse usually becomes small and irregular both in frequency

and force. It may assume the characters of *pulsus paradoxus*, becoming weak or disappearing altogether with inspiration. The embarrassment of the heart action by the effused liquid develops a tendency to syncope on exertion or emotional excitement; hence the patient avoids any movement. Generally he lies upon his back with the head slightly raised, or he may incline to the left side. In some instances the embarrassment of the circulation is so great as to induce orthopnœa. Dyspnœa is a common and important symptom. The expression of the patient is anxious, the face is dusky, and he feels a sense of impending danger. As the cardiac failure increases the venous circulation becomes more embarrassed, the cyanosis more marked, and a serous effusion is likely to occur in the abdominal and pleural cavities. General anasarca results in some cases, but more commonly the infiltration is confined to the extremities.

Among the more prominent pressure effects are distention of the veins of the neck—there may be a venous pulse—dysphagia, and an irritative cough. Compression of the left lung increases the dyspnœa. Aphonia is present occasionally from compression of the recurrent laryngeal nerve.

The onset of pericarditis may be announced by disturbances of the nervous system, or these may occur later in the disease. In mild cases there may be only headache, dizziness, and perhaps restlessness, while in severe cases there may be sleeplessness or delirium, which may be low and muttering or violent, requiring restraint. Melancholia with an inclination to suicide may supervene. Severe nervous symptoms are especially likely to occur when there is hyperpyrexia, and are probably due to the disease with which the pericarditis is associated, rather than to the pericarditis itself.

Physical Signs.—Since the symptoms of pericarditis are so vague and indefinite, recourse must be had to the physical signs to establish the diagnosis; but it must be remembered that not every case of pericarditis is sufficiently acute or diffused to be recognized clinically, and that many cases do not progress to the formation of large effusions. Hence in the following account of the physical signs a more or less typical case of pericarditis must be kept constantly in view. For convenience of description it is thought advisable to divide it arbitrarily into three stages—the dry stage, which embraces the fibrinous exudation, the stage of liquid effusion, and the stage of absorption.

Inspection of the chest tells little until the first stage of pericarditis is passed, unless it be the violent beating of the heart against the chest wall, and even after the stage of effusion has been reached the amount of information to be derived depends upon the elasticity of the ribs and their cartilages. In children and in young subjects distention of the pericardium shows itself by an arching forward of the præcordium and its neighborhood, with widening of the intercostal spaces, which may extend from the second to the sixth rib. There will be a restriction of the respiratory movements in this region, and if the effusion be large the whole of the left chest may remain stationary. There may be a prominence of the epigastrium from pressure upon the left lobe of the liver. If the chest wall has lost its elasticity or if old pleuritic adhesions are present binding it down, there may be only widening or bulg-

ing of the intercostal spaces in the præcordial region, or there may be no evidence at all upon inspection. Sometimes the apex beat is carried to the left and upward from its normal position, or a wavy motion may be visible over the heart. This lifting of the impulse is effected without any alteration in the relative position of the heart, and is simply a mechanical phenomenon, the accumulated liquid forcing the tip of the heart back from the chest wall, while a portion nearer the base strikes it. As absorption sets in, the chest will be seen to regain its normal shape, and there will be a return of the respiratory movements. Œdema of the chest wall is mentioned by some authors, but it rarely occurs independently of more or less general anasarca.

Palpation.—In the early stage of the disease the cardiac excitement is readily detected by applying the hand to the chest. A friction fremitus may be felt. The distinctness of the fremitus will vary with a change in the position of the patient, being most marked when the body is bent forward. This occurs because the heart has a greater specific gravity than the liquid by which it is surrounded, and tends to sink. The presence or absence of tenderness in the præcordial or epigastric region may be determined by palpation. As the liquid accumulates the fremitus will become less and less apparent. If the effusion be large, it will become imperceptible. Shifting the body from side to side will cause a change in the situation of the apex beat. Occasionally an undulatory motion will be communicated to the hand even when not visible. Palpation may be of aid in determining the shape of the pericardium and its degree of distention.

With absorption of the liquid effusion and reapposition of the roughened surfaces the friction fremitus will be evident again; the apex beat will become perceptible, will gradually assume its normal characters, and return to its former position.

Percussion.—The area of præcordial dulness is unaltered at first, but as the liquid accumulates it is found to increase both vertically and laterally. Its shape is that of the pericardial sac, irregularly conical, with its base on a line a little above the ensiform cartilage. Rotch and Ebstein have called attention to the importance of dulness in the fifth intercostal space to the right of the sternum in the early recognition of pericardial effusion. According to their observations, dulness first appears at this point, which Ebstein calls the cardio-hepatic angle, and is quite or nearly absolute, whereas the liver dulness in health is relative. Increase in the quantity of effused liquid causes a corresponding increase in the area of præcordial dulness. It may extend at times from the second rib or even the clavicle to the ensiform cartilage in the vertical direction, and from nipple to nipple laterally. The dulness extends to the right of the sternum in all large effusions. Pins states that in the pericarditis of very young children with only a moderate amount of effusion there is dulness over the left half of the back, which disappears in the knee-chest position. He attributes this dulness to the relatively large heart and small thorax of young children, and attaches much diagnostic significance to it.

Auscultation affords the first positive indication of pericarditis, and it is during the first stages of the disease that this sign is of greatest value. Hence an early diagnosis may be made in the major-

ity of cases. If the ear or the stethoscope be applied over the pericardium, friction sounds, produced by contact of the roughened pericardial surfaces, will be heard. They are rubbing, grazing, or creaking in character, compared at times to the creaking of new leather. The sounds are superficial and are restricted to the præcordial region. There may be only a single sound, especially when the inflammation is circumscribed and in the region of the great vessels at the base of the heart. When near the apex it is likely to be double and to possess the to-and-fro character ascribed to it by Watson. The friction sounds may be synchronous with the heart sounds or occur independently of them. As a rule, they will be heard with greatest intensity at the junction of the fourth rib with the sternum on the left side, but occasionally they are present only over the base of the heart. Under such circumstances the inflammation is limited to the neighborhood of the great vessels. The intensity of the sounds will be increased by bending the body forward, by a full inspiration, and by pressure upon the præcordium. It is effected by bringing the pericardial surfaces into closer contact. The fibrinous exudation may be abundant, and yet from softness of the fibrin or from weakness of the heart muscle no sounds will be audible. The heart sounds are normal or the second sound may be accentuated.

As the liquid accumulates the friction sounds become less and less distinct until they finally disappear. The respiratory murmur is no longer heard over the præcordial space, and the heart sounds become muffled and indistinct. The muffling of the heart sounds is very important in reaching a diagnosis. They become indistinct from the apex upward, but according to DaCosta the second sound rarely ever disappears. As absorption proceeds the heart sounds regain their normal characters and the friction sounds reappear. They remain until the disappearance of the fibrin or until adhesion takes place between the two pericardial surfaces.

Sequelæ.—These relate chiefly to the anatomical changes in the heart wall and structures surrounding the pericardium which are the direct or indirect results of the inflammatory action. When there has been a mild grade of inflammation the parenchymatous degeneration of the superficial layers of the heart muscle is quickly repaired. In severe cases the weakening of the muscle leads to dilatation, with subsequent hypertrophy of varying degrees, or if the inflammation is long continued there may be a connective tissue increase, crowding out of the muscle fibres, and a resulting cardiac fibrosis of greater or less extent. Circumscribed adhesions are harmless. Occasionally the pericardial cavity becomes obliterated from extensive adhesions. It is stated by some authorities that the heart is always left in a weakened condition by an attack of pericarditis. If the pericarditis be long continued, the heart may become so weakened that venous stasis ensues, with its train of symptoms. Congestion of liver, stomach, spleen, and kidneys occurs. The amount of urine decreases. Hydro-thorax and general anasarca are often observed.

Diagnosis.—Cases of pericarditis differ greatly in extent, duration, and severity. There are those in which the inflammatory action is confined to a limited portion of the pericardial surface, and when so limited the base of the heart is the seat of election: it may be that the great

vessels alone are involved; or the inflammation may affect only the apex. As before stated, the detection of pericarditis depends upon its physical signs. The diagnosis cannot be made from the symptoms alone. In diseases with which pericarditis is most often associated, such as rheumatism and the exanthemata, it is the duty of the physician to examine the heart frequently. Rigors or a distinct chill, followed by pain in the præcordial region, should direct attention at once to the heart. If the pain is severe, it may radiate down the left arm. It is increased by pressure and sometimes by a full inspiration. Instead of distinct pain there may be only a sense of uneasiness in the præcordium. Pain is commonly regarded as the most important symptom of pericarditis, yet it may be absent throughout the disease.

Hyperpyrexia usually ushers in pericarditis in acute articular rheumatism, though there may be a distinct lowering of temperature. The onset of pericarditis may be so insidious that there will be no symptom to draw attention to the heart until effusion occurs and produces dyspnœa or embarrassment of the circulation.

The symptoms may be referred entirely to the nervous system. Headache and restlessness occur in the milder cases, while in the severer ones there will be evidence of intense cerebral disturbance. Delirium, low and muttering or severe and requiring restraint, frequently appears. Melancholia with suicidal tendencies comes on at times, and may last from two weeks to as many months.

The importance of dulness in the fifth right intercostal space in the early recognition of pericardial effusion must be emphasized. Later on the shape of the area of præcordial dulness, the absence of or slight change in the outline in the recumbent posture, and the fact that the dulness does not extend to the back are presumptive signs of pericarditis. In young children, however, and in cases with large effusions, there may be dulness over the left back. Under such circumstances other signs and such symptoms as may be present must be taken into consideration. Pins states that in children this posterior dulness is due to atelectasis at the base of the left lung, and disappears in the knee-chest position. Pericarditis frequently complicates pneumonia and pleurisy, and the præcordial may be continuous with the lung dulness. If the anterior borders of the lung are bound to the chest wall, there may be but little change in the normal area of præcordial dulness even with large effusions. Pulmonary emphysema sometimes diminishes the area of præcordial dulness and may lead to a mistaken diagnosis. On the other hand, consolidation of the anterior borders of the lung may give rise to dulness which it will be very difficult to distinguish from pericardial effusion.

The presence of a friction sound which is superficial and limited to the region of the heart is diagnostic of pericarditis if the sound continues when respiration is voluntarily suspended. This statement, however, will not hold good for those rare pleuro-pericardial sounds produced by the movements of the heart against a roughened pleura. It will be impossible to make a diagnosis in these cases unless there be intermission of the sound during some of the heart beats. The friction sound in pericarditis is often limited to the base of the heart, and may continue after a moderately large effusion has occurred. Pericardial

friction sounds become indistinct from the apex upward as the effusion increases. Being dependent upon the separation of the two layers of the pericardium they never disappear suddenly, though they may be heard at one visit and not at the next, so rapidly does the liquid accumulate in some instances. Again, they may change their seat and character during the visits of the physician. As the effusion increases in quantity the heart sounds become muffled and indistinct. This muffling of the heart sounds is an important aid in diagnosis, and is most apparent at the apex.

The diagnosis of hemorrhagic pericarditis is presumptive in purpura and scurvy. Should there be evidence of septic infection in connection with the symptoms and signs of pericarditis, the presence of pus in the pericardium is probable. In such cases the previous history should be taken into consideration. Purulent pericarditis most often follows injury to the pericardium or suppuration in some adjacent structure. When there is doubt aspiration is justifiable. Pus is rarely absorbed and operative procedures are often indicated. But before operative interference is undertaken tuberculosis should be excluded as an etiological factor.

It now becomes necessary to differentiate pericarditis from the diseases for which it is most likely to be mistaken.

A question of differential diagnosis between pericarditis and *endocarditis* can arise only in the first stage of the former disease. As soon as effusion occurs there can be no doubt, though it must be remembered that the two affections may coexist. The following distinctions will hold good whether the endocardial murmurs have origin in anæmia or in inflammation of the endocardium. In ordinary cases the friction sounds of pericarditis may be distinguished from endocardial murmurs by their superficial character, by their rougher quality, and by their limitation to the præcordial area. Pericardial friction sounds have a rough, grating quality as a rule, and appear to be immediately under the ear. Endocardial murmurs are soft and blowing. Yet pericardial sounds sometimes possess these qualities. In such cases it is often impossible to make a diagnosis.

Pericardial sounds are confined to the præcordial area, occasionally to a definite portion of it, and have their greatest intensity at the junction of the fourth rib with the sternum on the left side. Endocardial murmurs are conveyed beyond the limits of the præcordium, to the right or left, along the course of the vessels in the neck, and sometimes to the back. A pericardial friction sound may change its seat or character, while an endocardial murmur never does. The intensity of a pericardial friction sound may be altered by change in position of the patient. Bending him forward will increase the intensity by bringing the pericardial surfaces into closer contact, while bending him backward will diminish it. Moreover, the intensity may be increased by a full inspiration. Endocardial murmurs are not thus affected; they are often decreased by full inspiration. Pericardial friction sounds bear no definite relation in time to the heart sounds. They may be double, or, as Osler states, possess a canter rhythm. Endocardial murmurs precede, take the place of, or follow the heart sounds. Often they entirely mask them.

Pleurisy sometimes simulates pericarditis, not only in its symptoms, but in its physical signs. The dry, irritative cough and dyspnœa occur in both diseases, but the physical signs differ in location. When pericarditis complicates pleurisy or pneumonia it is often overlooked. Pleurisy does not give rise to bulging in the præcordium. The friction sound of pleurisy is likely to be confounded with that of pericarditis only when it is confined to the præcordial region. In such cases voluntary suspension of respiration will cause the pleuritic sound to cease. The difficulty of distinguishing a pericardial from a pleuro-pericardial friction sound has been referred to. If the sound intermits during one or two beats of the heart, it may be considered of pleuritic origin. The shape of the præcordial dulness and the fact that except in large effusions it does not extend beyond the præcordium will serve to distinguish the two diseases. In pleurisy dulness is present over the whole of the left chest, being most marked in the back. If there be dulness posteriorly in pericarditis, it may disappear in the knee-chest position. Perfect distinctness of the heart sounds in pleurisy is another point in diagnosis.

Hypertrophy of the heart may be mistaken for pericarditis in the stage of effusion, because of the increased area of præcordial dulness, yet a careful inquiry into the physical signs will suffice to differentiate the two conditions. In hypertrophy the force of the impulse is increased, and, if it is displaced, it is carried to the left and downward. In pericarditis the force of the impulse is diminished, and it is displaced upward and to the left. In pericarditis the abnormal area of dulness extends to the left of the apex beat, often as much as two inches, which is never true in hypertrophy. Such an extension of dulness beyond the apex beat may be considered diagnostic of pericarditis. In hypertrophy the heart sounds are intensified, while in pericarditis they are muffled and indistinct, and may even be inaudible at the apex. If dilatation coexists with the hypertrophy, the diagnosis may present greater difficulty. The impulse is wavy and diffused in dilatation, the area of dulness is more or less quadrilateral rather than triangular as in pericarditis, and there is only a relative dulness in the fifth right interspace. When the dilatation is advanced and the heart sounds are weak, it may be impossible to make a differential diagnosis between dilatation and pericarditis.

Tumors of the mediastinum sometimes simulate pericarditis. Flint has recorded a case in which the heart was enveloped in a neoplasm which filled and distended the pericardial sac without altering its shape. In such a case it is impossible to make a differential diagnosis. New growths in the mediastinum may give rise to præcordial dulness, may cause displacement of the apex beat and interference with the heart's action, and may cause pressure effects upon surrounding structures. But the dulness they produce is not uniform in outline, and varies with the situation of the tumor; moreover, the area of dulness is rarely triangular, such as we find in pericarditis. The displacement of the apex beat varies in different cases, and if a solid tumor lies between the heart and chest wall, the heart sounds may be intensified.

DaCosta calls attention to the fact that pericarditis may assume the characters of *gastric irritation* or *inflammation*. There will be nausea

and vomiting, and tenderness in the epigastric region. An examination of the præcordium will establish the diagnosis.

Prognosis.—The prognosis in pericarditis varies with the intensity and extent of the inflammation and the nature and severity of the disease with which it is associated. The age at which pericarditis occurs influences the prognosis to a great extent. It is very fatal in children and in the aged. The prognosis in traumatic pericarditis will depend upon the extent and character of the injury. A guarded prognosis should be given until it can be determined whether the effusion will become purulent. A low grade of circumscribed pericardial inflammation tends of itself to recovery, but if the inflammatory action be diffused and of a severe type, the prognosis is grave. Sudden and intense pericarditis usually terminates fatally. The rapidity with which the effusion takes place influences the prognosis to a greater extent than the amount of the liquid. Sudden death may occur in a few hours from the onset of the disease from compression and paralysis of the heart. Pericarditis complicating acute articular rheumatism ends in recovery in a majority of cases. The prognosis is unfavorable when marked hyperpyrexia attends its development. In connection with nephritis, on the other hand, the majority of cases terminate fatally. The prognosis is exceedingly grave when pericarditis is associated with a severe pneumonia or pleurisy. As a rule, the prognosis is good in pericarditis complicating the exanthemata. The development of marked nervous manifestations does not of necessity add to the gravity of the disease. The prognosis is unfavorable in cases where there is reason to believe that myocarditis accompanies the pericarditis. In rendering a prognosis the character of the exudation must be taken into consideration. Death usually supervenes when it is hemorrhagic or purulent. The success, however, attending operative interference in purulent cases renders the prognosis more favorable than formerly. Pus is rarely absorbed. Tubercular pericarditis almost always ends fatally sooner or later. Occasionally acute pericarditis passes into the chronic form, or, rather, is accompanied by a large effusion which disappears slowly. Relapses are likely to occur, and thus the disease drags on for months. As a result of the long continuance of the effusion the heart muscle undergoes extensive degeneration, its propelling power is diminished, and dilatation ensues. During the progress of the disease the patient suffers from repeated attacks of extreme dyspnœa, and death may take place from sudden syncope or œdema of the lungs. Any sudden effort may cause instant death. If recovery follows dilatation of the heart, compensatory hypertrophy is developed.

When adhesions form the prognosis will vary according to their extent and situation. Circumscribed adhesions may give rise to no symptoms. Extensive adhesion of the pericardial surfaces is followed by dilatation, and the heart wall is left permanently weakened. Adhesions about the base are likely to interfere with the coronary circulation and lead to cardiac atrophy or fibrosis.

Treatment.—The most important part of the treatment of pericarditis is absolute quiet, both of body and mind, and especially is this true after the stage of effusion has been reached. The least exertion or emotional excitement may induce fatal syncope. Under no circum-

stances should the patient get out of bed, and all persons who are not necessary to the welfare of the patient should be kept from the room.

Since pericarditis may arise from so many different causes, the line of treatment adopted will vary somewhat with the individual requirements of each case.

In the early stage of pericarditis an attempt should be made to control the inflammatory action. Opinion is divided as to whether hot or cold applications are the more efficacious. The French and German schools advocate the use of cold, applied in the form of an ice bag, changed as often as may be necessary, or Leiter's coil. They contend that the intensity of the inflammation is lessened, that the heart action is steadied, and that the pain is relieved. Many patients, however, cannot endure the application of cold, and with them hot anodyne poultices must be substituted.

The diet should be light and nourishing, consisting largely of milk and eggs. Beef, mutton, and chicken broth may be given at intervals if desired, but it must be remembered that they are stimulants rather than foods.

In sthenic individuals the frequency and force of the heart-action may be controlled by veratrum viride or aconite, but their action must be closely watched, and must never be carried to actual depression. The treatment of pericarditis should be supporting rather than depressing or depleting. For this reason general bloodletting has been abandoned, and local bloodletting is admissible only in sthenic cases. The application of ten or twelve leeches to the præcordium is often followed in these cases by marked relief; the pain diminishes and the heart becomes more quiet.

Loomis states that opium is to be relied upon more than any other drug in the first stage of the disease, and advises the use of small doses, repeated as often as may be necessary to relieve the pain, steady the heart's action, and quiet the patient. Restlessness is especially likely to manifest itself or to increase at night, and opium affords the patient quiet and sleep. It may be administered in the form of powdered opium, Dover's powder, or as morphine hypodermically. It is never advisable to bring the patient into a state of semi-narcotism. Chloral has been advocated as a substitute for opium, but is objectionable on the ground that it is a cardiac depressant.

Hyperpyrexia should be treated by sponging the body and limbs with tepid or cold water, or, if necessary, by the cold pack. Internal antipyretics must be avoided, because of the cardiac depression they produce. If in acute articular rheumatism treatment with the salicylates—and this applies equally to salol and oil of wintergreen—has been instituted, they must be discontinued as soon as the pericarditis is discovered. DaCosta states that they are not only useless as remedial agents, but may be productive of actual harm from their depressing action on the heart. The alkaline treatment should be substituted.

When pericarditis occurs in the course of septicæmia or pyæmia free stimulation is called for early. The same is true if there is great restlessness or cerebral excitement, unless the alcohol is poorly borne. Whiskey (Scotch) is to be preferred—brandy or wine may be given and in sufficient quantities to support the patient. In any case alcohol may

be prescribed as freely as the requirements indicate. When the heart action is turbulent, digitalis in the form of the tincture or infusion is of service in reducing the frequency and increasing the force of the contractions.

As the liquid effusion accumulates and the heart becomes embarrassed, stimulation with alcohol or digitalis should be pushed until its action is under control. If this should fail and there is imminent danger of paralysis of the heart from compression, paracentesis of the pericardium should be resorted to *without delay*. The relief afforded by this operation is often marked. The question of the performance of paracentesis in any case concerns not so much the actual quantity of liquid effused as the rapidity with which the effusion takes place, since a relatively small quantity rapidly effused may cause such cardiac, and secondarily such respiratory, embarrassment as to demand immediate surgical interference. According to DaCosta, when pericarditis is complicated by pleurisy with effusion, the dyspnœa, which in reality is cardiac, may be relieved at times by tapping the pleural cavity.

The point usually selected for the insertion of the aspirating needle is the fifth left interspace, about two inches from the median line of the sternum. Special instruments have been devised by Roberts and Pepper for tapping the pericardium, with the idea of preventing wounding of the heart, but an ordinary aspirating needle, provided it be sufficiently large, will answer the purpose in the majority of cases. Since the heart, as a rule, is forced back from the chest wall by the accumulated liquid, and since it tends to sink farther with the patient in the recumbent posture, there is but little danger of wounding it until the greater part of the liquid has been drawn off. Should the needle penetrate the ventricular wall in any case, the accident will be made known immediately by violent moving of the needle. Such an accident is not necessarily attended by any untoward effect. The ventricles have been entered and blood withdrawn without the production of harm. It must be mentioned, however, that death has occurred from tearing the thin wall of the right ventricle with the point of the needle. Hence it is advisable to enter the pericardial cavity only so far as may be necessary to draw off the liquid. This may be determined readily by drawing on the piston of the aspirator as soon as the point of the needle is well under the skin, and preserving the vacuum thus formed until the liquid is reached. Surgical cleanliness is necessary in performing the operation, for fear of inoculating the pericardial cavity with pyogenic organisms, and converting a sero-fibrinous into a purulent effusion. Before introducing the needle the chest wall should be thoroughly disinfected. The skin must be first washed with soap and warm water to remove any fatty matters that may adhere to it, then with alcohol, and lastly with ether. After the liquid has been withdrawn and before the needle has been removed a piece of rubber plaster should be fixed by one side to the chest wall in position to cover the site of puncture as soon as the needle is removed. In case the effusion reaccumulates repetition of the operation may become necessary.

The absorption of any effusion which has not demanded paracentesis may be hastened by the use of diuretics—potassium acetate or potassium citrate, combined with the infusion of digitalis—and hydragogue

cathartics—sodium phosphate, sodium sulphate, sodium and potassium tartrate, etc. But these drugs, and especially the potassium salts, must be used with care, for both potassium and sodium are cardiac depressants. The patient is in a weakened condition and the heart is already depressed by the disease. Calomel in small doses is to be recommended in certain cases. Occasionally when absorption is long delayed removal of a portion of the effusion by aspiration will hasten the disappearance of the remaining liquid by natural means. In these cases it is supposed that the tension on the pericardial sac closes the lymphatic channels, as in pleurisy with effusion, and that when this is taken off absorption proceeds normally. Potassium iodide in doses of 40 grains a day is said to be of service in the promotion of delayed absorption. Blisters to the præcordium are recommended for the same purpose.

Quinine in tonic doses—4 to 6 gr. a day—and iron are indicated in this stage. As soon as the patient's condition will permit a more generous diet should be allowed, including the more easily digested meats.

Purulent pericarditis demands the free opening of the pericardium, washing out, and draining. For the details of the operation the reader is referred to works on surgery.

Chronic Pericarditis.

The line of demarcation between acute and chronic pericarditis is not sharply defined. Acute cases merge imperceptibly into the chronic form. As a general rule, it may be stated that a case of pericarditis which extends over a period of three or four weeks without progressing toward recovery or ending in death has become chronic.

Chronic pericarditis may result from the same causes which are operative in the production of the acute variety. It may follow the acute, or may be subacute or chronic from the outset. Tubercular, cancerous, and purulent pericarditis is sometimes of a chronic nature from the beginning. Tubercular pericarditis may extend over a period of a year or more. When a tubercular pericarditis has sero-fibrinous inflammatory characters added to it, if these are extensive, acute symptoms manifest themselves.

Chronic pericarditis may occur with or without effusion. When an effusion is present it may accumulate to a very large amount. Chronic tubercular pericarditis is often characterized by great thickening of the pericardium and followed by dense adhesions. A purulent effusion may become inspissated and be the seat of calcareous deposit. Such calcification may be limited to the base of the heart or entirely surround it, and give rise to the condition which was formerly called "bony heart."

Extensive adhesions between the two layers of the pericardium are not infrequently found at the autopsy table in cases where no history of a previous pericarditis could be elicited. These cases were probably chronic from the outset and gave rise to few, if any, symptoms.

Many of the "milk patches" so commonly seen are undoubtedly the result of chronic circumscribed pericarditis rather than the acute variety. The symptoms, physical signs, diagnosis, and treatment of pericarditis have been discussed at sufficient length in the foregoing article.

Adherent Pericardium.

Adherent pericardium is a sequel of pericarditis, and results from organization of the fibrinous exudate, the pathology of which has been given (see page 359). The adhesions vary in appearance and extent. They may be slender bands, relatively long, or they may be short and thick. Large or small areas of the visceral or parietal layers of the pericardium may be adherent, and in extreme cases there is entire obliteration of the pericardial cavity.

Undoubtedly, the adhesions in many cases of pericarditis become absorbed after varying lengths of time, and the only evidence of them at the autopsy table, if any be present, is the "milk patches" so frequently found. Slender adhesions are more likely to be absorbed than thick ones, and in any case bands are more likely to be absorbed than agglutinated surfaces to separate. The disappearance of connective tissue bands is brought about mainly by the action of the heart, constantly dragging upon them and interfering with their nutrition. Furthermore, the very contraction of the connective tissue as it loses its embryonic characters causes obliteration of its vessels and renders the lymphatic circulation more difficult.

Pericarditis is complicated at times by inflammation on the outer surface of the pericardium and its extension to adjacent structures. These cases, called mediastino-pericarditis, may end not only in the formation of adhesions between the two layers of the pericardium, but also between the outer surface of the pericardium and neighboring structures. The lung may become fixed to the pericardium to the side of or in front of the heart, or the anterior surface of the pericardium may be bound to the chest wall.

The most extreme thickening of the layers of the pericardium is met with in chronic tubercular pericarditis.

Adhesions do not cause secondary changes in the myocardium unless they are sufficiently extensive to interfere seriously with the heart's action or in position to impede the coronary circulation. There seems to be some difference of opinion among observers as to whether hypertrophy and dilatation or atrophy follows adherent pericardium. The occurrence of the one or the other condition would seem to be governed, at least in part, by the nature and situation of the adhesions. Were the adhesion simply to increase the amount of work the heart had to perform by tending to hold back the ventricular wall during systole, hypertrophy with subsequent dilatation would naturally follow. If, on the other hand, the bands of adhesion were so situated as to interfere with the coronary circulation, atrophy, or perhaps cardiac fibrosis, would result. Such an explanation harmonizes the apparent discrepancies found in the writings of the different authors on this subject. As a matter of fact, either condition may be met with—hypertrophy and dilatation or atrophy, the former being the more common.

The symptoms of adherent pericardium are ill defined and not to be relied upon. Symptoms which are said to be characteristic are found in cases which at the autopsy reveal no adhesions, and, on the other hand, extensive adhesions have been found in cases which presented no symptoms during life. The conditions which determine the manifesta-

tion of symptoms have to do chiefly with secondary changes in the heart itself, and are in no way characteristic of adhesions. Even the extent of the adhesions does not always influence the character or the severity of the symptoms. It may be safely said, however, that extensive adhesions are more likely to give rise to symptoms than are those which are circumscribed, and circumscribed adhesions rather than bands of connective tissue between the two layers. In general terms, it may be stated that adhesions which interfere with the heart's action or with the coronary circulation sooner or later give rise to manifestations, and the more serious the interference the sooner do they present themselves and the severer they are.

The *pulsus paradoxus*, which is characterized by an increase in frequency of the pulse during inspiration, but a diminution in volume until it becomes, in certain cases, imperceptible at the wrist, was formerly considered pathognomonic of adherent pericardium, but is now known to occur in other conditions.

The PHYSICAL SIGNS of adherent pericardium are more pronounced than the symptoms, but even they are not reliable. Such physical signs as are dependent upon hypertrophy or dilatation will be considered under their appropriate headings (pages 416, 424). The physical sign upon which the greatest amount of dependence is to be placed is retraction of the chest wall during systole of the heart. The retraction may be determined at times by palpation when it is not visible, and is usually more marked on inspiration. It is believed, however, that this sign is present only in cases where the pericardium is adherent externally to the chest wall. Such retraction is most frequently seen in the region of the normal impulse or at the xiphoid cartilage. Following the retraction there is oftentimes a rebound or shock against the chest wall, with the occurrence of diastole, which is called the diastolic shock. In other cases the visible impulse will be irregular and is described as "jogging."

Attention was first directed to collapse of the jugular vein during diastole by Friedreich. It is sometimes spoken of as "Friedreich's sign." When adherent pericardium exists there is little or no change in the position of the cardiac impulse with change in the position of the patient. A feeble impulse at the apex, while the body of the heart strikes the chest wall a little higher up, is presumptive evidence of adherent pericardium, provided effusion is excluded.

There is but little to be discovered upon percussion unless hypertrophy and dilatation or atrophy coexist with the adhesions. Williams states, however, that the area of cardiac dulness is fixed upward and to the left, and is not altered during deep inspiration. Cardiac hypertrophy in children or young adults without definite cause for its occurrence renders the presence of pericardial adhesions probable. Auscultation gives evidence only of coexisting murmurs or changes in the heart sounds dependent upon hypertrophy or other abnormal conditions. Riess has noticed a metallic character in the heart sounds in cases where adhesions pull upon the stomach and introduce its resonance as a disturbing factor.

ENDOCARDITIS.

By ALFRED LEE LOOMIS, M. D.

DEFINITION.—Endocarditis is the term employed to designate inflammation of the endocardium. Such inflammation is generally limited to the valvular endocardium, and has received the name of valvular endocarditis. It is usually described as *acute* and *chronic*, but the acute so often merges imperceptibly into the chronic that it is difficult, and at times impossible, to determine when a case ceases to be acute and when it becomes chronic. It is well to recognize three distinct varieties of endocarditis—the *simple*, the *malignant*, and the *fibrotic*. The simple and malignant are usually acute, the fibrotic is always chronic.

SIMPLE ACUTE ENDOCARDITIS.

ETIOLOGY.—Simple acute endocarditis is associated with a variety of infective processes, though it is met with most frequently in the course of acute articular rheumatism. The majority of cases of acute articular rheumatism are complicated by endocarditis during the second week of the rheumatic attack. Sometimes it precedes the articular manifestation of the rheumatic infection. Chorea in one who has a distinct rheumatic history is often associated with endocarditis. Occasionally it complicates acute tonsillitis. It is liable to occur in the course of any of the acute infectious diseases. I have met with it, however, more often in scarlet fever than in any other of the exanthemata. It is an occasional complication in the specific form during convalescence. In estimating the etiological importance that any disease bears in the production of endocarditis it must be remembered that not every blowing sound or murmur is indicative of an inflamed endocardium.

Simple acute endocarditis is rarely met with in diphtheria, pneumonia, pyæmia, or septicæmia; the endocarditis met with during the course of these diseases is more often malignant. In acute Bright's disease complicated by pericarditis simple acute endocarditis is almost always present. The specific micro-organism of simple acute endocarditis has not yet been detected, although the specific bacteria of the diseases in which it occurs are usually present in the endocardial changes, and are undoubtedly etiological factors in its development.

It may be questioned if age has any distinct predisposing influence in the production of this disease, although the diseases with which it is most frequently associated are diseases of childhood rather than of adult life. Consequently, it is met with more often during the early period of life. The endocardium of the right heart is usually the seat of the disease during fœtal life, and that of the left heart after birth.

Pathological Anatomy.—The anatomical changes of simple acute endocarditis consist in proliferation of the endothelial connective tissue elements and round cell infiltration in the subendothelial tissue. On the surface of the inflamed part fibrin is deposited, the superficial elements having undergone necrosis. These fibrinous deposits give rise to minute vegetations which rarely exceed 4 mm. in size. They are regarded as the characteristic lesion of this form of endocarditis, but they are nothing more than granulation tissue covered with fibrin. Micro-organisms may be found at the site of these deposits; their significance has not yet been determined.

These vegetations may increase in size and become the seat of ulceration, or they may be absorbed and the endocardium become thickened. In rare instances they become detached, giving rise to emboli which cause infarctions in different organs. In the majority of instances permanent lesions of the valves result from fibrotic changes. It is this tendency to fibrosis which gives to simple acute endocarditis its greatest danger. The mitral valve is more often the seat of these changes than the aortic. The changes in the mitral valves are for the most part on its auricular surface, while the changes in the aortic valve are on the ventricular surface; in other words, these vegetations are found chiefly on the surface of the valve which is opposed to the current of the circulation. They are usually arranged on the borders of the aortic valves a little distance from their edges. In the centre of the band of tissue which passes from the attachment of the valve to the Arantian body the granulations are most numerous. Upon the auricular surface of the mitral valve near the insertion of the tendons the irregular wreaths of vegetations enclose the attachment of the chordæ tendineæ. The tendons themselves may become so soft and friable as to rupture, or they may become adherent to each other. When such adhesions occur, either with agglutination of the flaps to each other or to the heart walls, mitral incompetency or stenosis may result. It is to be remembered that the principal danger in all cases of simple acute endocarditis is from the fibrotic changes which take place in the valves and the valvular orifices.

Symptoms.—Simple acute endocarditis in the majority of instances is unattended by cardiac symptoms. Its advent is so insidious, and its symptoms and physical signs are so uncertain, that in its early stages one always hesitates to state positively that it exists. The symptoms which excite suspicion are a sense of distress sometimes amounting to pain in the præcordial region. In children the pain is often referred to the epigastrium and is accompanied by vomiting. The heart's action is increased in frequency during excitement or after physical exertion. Often it becomes irregular and is accompanied by palpitation. The pulse becomes feeble and sometimes dicrotic. The patient is restless and anxious and prefers a half-sitting posture. A hacking cough may be present, accompanied by slight dyspnœa. Variations in temperature are not constant or significant. A slight rise in temperature, however, is often the only symptom of an acute endocarditis complicating chronic valvular disease, and when the rise in temperature persists in such a condition for a number of days without any other assignable cause, it is a strong diagnostic point. The duration of simple acute endocarditis

varies from two to six weeks. In the majority of cases convalescence is established in four weeks.

PHYSICAL SIGNS.—In the early stages *inspection* and *palpation* may show an increase in the force and rapidity of the cardiac impulse; later the force of the impulse is diminished. There is nothing, however, diagnostic either in these changes or in the changes in the area of the *percussion* dulness. The most significant, but by no means certain, physical signs of simple acute endocarditis are adventitious sounds or murmurs which replace the normal heart sounds. Blowing sounds accompanying or taking the place of the heart sounds which are not due to endocarditis, but to the attendant blood changes and muscular incompetency, are, however, so frequently heard in acute rheumatism and other febrile diseases that the occurrence of a murmur in any of these conditions cannot be regarded as certain evidence of endocarditis. If, however, during the first two weeks of acute rheumatism a murmur is developed which has been preceded for some time by a prolongation of the first sound of the heart, the probability is that the endocardium is affected. Usually under such circumstances there will be other evidences of disturbance of the circulation, such as palpitation accompanied by distinct interference with the force and rhythm of the heart's action. It is to be remembered that a murmur alone is a very unreliable sign of endocarditis. I have occasionally noticed, two or three months after recovery from an attack of acute rheumatism in vigorous adults, that murmurs and cardiac symptoms would develop, which left no doubt that an endocarditis had accompanied the rheumatic attack, though it had not been attended by a murmur during its acute stage.

DIAGNOSIS.—While the diagnosis in a large proportion of cases of simple acute endocarditis must always be uncertain, in a number of instances where the evidences of the existence of endocarditis are well established it is difficult to determine whether it is simple or malignant. Ordinarily, the history of the case and the renal and other visceral complications are sufficient to indicate a malignant endocarditis. There are, however, cases of malignant endocarditis which are so mild in type that one does not suspect their malignant character until an unexpected fatal issue reveals their true nature. Simple acute endocarditis sometimes, on the other hand, occurs in conditions which simulate the malignant and lead to error in diagnosis.

COMPLICATIONS AND SEQUELÆ.—Simple acute endocarditis is in rare instances complicated by embolism and the development of infarctions. When pleuro-pneumonia complicates this variety of endocarditis it is usually of embolic origin.

PROGNOSIS.—A large percentage of cases of simple acute endocarditis terminate in fibrotic valvular changes. Complete recovery in rheumatic subjects is rarely met with, for the tendency to recurring endocarditis is very great, and there is always more or less thickening and induration of the valves remaining after the primary attack. I have never been able to demonstrate that cardiac fibrosis occurs as a sequel of endocarditis unless fibrosis of the coronary vessels existed prior to the endocardial changes.

TREATMENT.—The prophylactic treatment of simple acute endocarditis consists in counteracting rheumatic tendencies by a restricted diet,

moderate exercise in the open air, and close attention to the rules of general hygiene. Cod-liver oil should be administered daily to young subjects of this class, and flannel should be worn constantly next to the skin in order that the surface of the body may be protected from sudden variations of temperature. On the occurrence of the slightest rheumatic symptoms the individual should be kept in bed and his diet restricted to milk containing a small quantity of bicarbonate of soda. It is doubtful if the salicylates exert any prophylactic influence against this disease. My experience is strongly against their use.

When endocarditis is once established I have never found that drugs had any controlling influence over it. The temperature of the sick-room should be kept between 70° and 75° F., the patient restricted to a milk diet, and the urine kept as near neutral as possible. If the heart is irregular in its action and the patient restless, from $\frac{1}{20}$ to $\frac{1}{8}$ of a grain of the acetate of morphine should be administered at regular intervals, sufficient to keep the patient in a quiet, comfortable state. All applications over the præcordial space, except such as will maintain an equable temperature of the surface, have seemed to me to be not only useless, but harmful, especially cold applications. It is to be remembered that this inflammation rarely subsides within four weeks, and this course therefore should be rigidly enforced during that period. Too great care cannot be exercised during convalescence to protect patients against exposure to changes of temperature and excessive fatigue. A careful regulation of the diet must be insisted upon, and iron and cod-liver oil in small quantities must be taken constantly after meals.

Malignant Endocarditis.

Malignant endocarditis is also known by the names ulcerative, diphtheritic, and infectious.

Etiology.—Primary malignant endocarditis in persons who are in perfect health at the time of the attack is of rare occurrence. Two typical cases of malignant endocarditis were recently admitted to my wards in Bellevue Hospital that were evidently of primary origin. One died in twenty-four hours, and the other on the third day after admission. The only lesions found at autopsy were those of malignant endocarditis. Those are the only cases of this affection that have come under my observation that were evidently not secondary to some septic process or were not associated with, or secondary to, some acute infectious process. The infectious disease with which malignant endocarditis is most often associated is pneumonia. Such cases are often complicated by pericarditis and meningitis. This form of endocarditis may be a complication of the exanthematous fevers. It may be secondary to old valvular disease or to some form of valvular traumatism.

Pathological Anatomy.—The valves and the chordæ tendineæ of the left heart are the usual seat of this form of endocarditis; in some instances the endocardium lining the ventricular cavity is involved. Its characteristic lesions are vegetation, ulceration, and suppuration of the valves. The vegetations vary in number as well as in size. They are usually of a greenish yellow color. The ulceration may be either superficial, involving only the endocardium, or deep seated, involving

the deeper structure of the valves. Acute multiple abscesses which develop into ulcers are sometimes found at the base of the vegetations. The ulcers, however, formed either by necrosis of the vegetation or by multiple abscesses, are irregular in shape, with everted edges and a gray base. When there is extensive loss of substance, perforation or rupture of the valves may occur. The perforations are sometimes closed or hidden by a fibrinous deposit. The vegetations are often torn into long shreds by the force of the blood current, and become a free mass capable of spreading the disease to other portions of the endocardium by contact, either to the walls of the heart or aorta. Valvular aneurysms often result from these destructive processes. A variety of micro-organisms are found associated with the destructive processes of this affection, the pyogenic micrococci most frequently. The pneumococci are usually found when it complicates pneumonia, and the gonococcus in those cases that occur with gonorrhœal rheumatic manifestations. It is claimed that a specific microbe is present in primary malignant endocarditis.

The vegetations and ulcerations which occur in this disease give rise to the most diverse lesions. Masses may be detached from diseased areas, and, having entered the current of the circulation, produce a variety of changes in the organs and tissues in which they are carried. It is to be remembered also that these emboli, whether of large or small size, carry infectious elements to the different organs and tissues. If they are of large size, they may obstruct large vessels; for instance, the femoral or external iliac may be obstructed and lead to gangrene of the extremity. Capillary embolism may occur in a number of organs at the same time. The infarctions which result from the emboli may be few in number and confined to one organ, or they may be numerous, scattered through different organs. The spleen and kidneys are the organs most often involved, but infarctions may develop in the brain, intestines, skin, or, in fact, in any tissue of the body. When the skin is involved ecchymotic spots are produced, followed by cellulitis. Cerebral emboli may lead to meningitis, softening or abscess of the brain. Pleurisy, parotiditis, and embolic gastric ulcer are sometimes associated with malignant endocarditis. Besides the local lesions which occur in this disease, the septic phenomena are important, such as the typhoid condition, acute jaundice, an intermittent fever, or renal symptoms. The infarcts which occur in this variety of endocarditis always tend to suppuration unless the associated endocarditis is secondary to an old valvular lesion which is free from any specific infection.

Symptoms.—In the majority of instances the cardiac symptoms are not prominent, and they may be altogether masked by the symptoms of the antecedent affections. There is usually an initial chill, followed by fever of an intermittent, remittent, or typhoid type, attended by great prostration, an unusually rapid pulse, and profuse perspiration. The spleen will at the same time be found enlarged and tender. As the disease progresses symptoms due to the development of infarction and secondary inflammation will become prominent. These will vary according to the organs or tissues that may be the seat of the embolic processes. Thus, the symptoms may be such as to indicate involvement of the kidneys, brain, lungs, intestines, or skin and subcutaneous tissue.

Clinically, one meets with two distinct types of this affection—(1) a typhoid and (2) a septic or pyæmic type.

(1) The *typhoid* type is characterized by a remittent fever with a temperature ranging from 101° to 107° F. throughout its active course. Its onset may be abrupt, ushered in by a distinct chill, or it may be gradual, resembling the onset of typhoid fever. Vomiting, diarrhœa, and a tympanitic abdomen with great prostration are early symptoms. Later, the pulse becomes frequent, often reaching 120; the tongue becomes brown and dry, sordes collect upon the teeth, a low muttering delirium comes on with subsultus tendinum, and the patient is almost constantly bathed in a profuse perspiration. A rose-colored or petechial eruption often makes its appearance, and the patient gradually passes into a condition of partial or complete coma. On examination the spleen will be found enlarged and tender, but there are few if any cardiac symptoms. In some cases the most careful examination will fail to detect a murmur; in others a systolic apical murmur will be heard, but the true significance of such murmurs cannot always be determined. I have met with two cases in which extensive valvular lesions were found after death, and no murmurs could be detected during life.

(2) The *septic* or *pyæmic* type is met with most frequently in connection with traumatism, suppuration, diseases of the bones, and puerperal sepsis. It is invariably ushered in with a chill, followed by a well-pronounced intermittent type of fever. The chills, fever, and sweats clearly resemble those which ordinarily attend pyæmic infection. In some cases the temperature may suddenly fall from 106° to 97° F., while the pulse will range constantly between 120 and 140. The skin becomes swollen, and occasionally the body is deeply jaundiced. The cardiac symptoms are as obscure and uncertain as in the typhoid type, and they may not be apparent until attention is called to them by the occurrence of embolic processes of a suppurative type or the appearance of a petechial eruption.

Now and then one meets with patients with old valvular lesions in whom symptoms are developed similar to those which occur in the typhoid or septic type already referred to. When a physical examination gives evidence of the development of an acute endocarditis, these cases may run either a very acute course or, if the endocarditis is not severe, a chronic one extending over months. I saw one case of a young girl, eighteen years of age, in whom the fever and embolic symptoms continued for five months. Infarctions occurred in nearly all the organs, and the skin and subcutaneous tissue almost over the entire surface were at some time involved. She finally died in a condition of extreme anæmia.

There are no distinctive physical signs of this form of endocarditis. A mitral systolic murmur must not be regarded as a sure indication of its presence, although when such a murmur is heard, varying in its intensity and position from day to day and accompanied by the general symptoms already referred to, there is strong presumptive evidence that this form of endocarditis exists, which is rendered almost certain if an aortic murmur becomes associated with it.

The DURATION and COURSE of this disease vary greatly in different

cases, influenced by the nature of the primary disease. Its usual limit is from four to six weeks; when it occurs in connection with old valvular disease it may continue for months.

Diagnosis.—The diagnosis of this form of endocarditis is often difficult. It may be confounded with *simple acute endocarditis*, *typhoid fever*, *septicæmia*, and *pyæmia*. Usually, however, it is readily distinguished from simple acute endocarditis by the presence of typhoid and pyæmic symptoms associated with signs of embolism. It differs from typhoid fever in its mode of onset and rapid course, in its range of temperature and pulse rate, and by the presence of symptoms indicating infarctions. Although in typhoid fever infarction sometimes occurs, it is only when the fever has continued for weeks in extremely debilitated subjects. Then the history of the typhoid case, prior to and during the early period of the fever, differs so essentially from malignant endocarditis that one would hardly make a mistake. In most instances the temperature range during the first week of typhoid fever will be sufficient to distinguish it from the irregular pyæmia of malignant endocarditis. Occasionally, however, the symptoms of these two affections simulate each other so closely that a differential diagnosis will be impossible, especially when typhoid fever is complicated by infarctions and parotiditis. There are no reliable points of differential diagnosis between malignant endocarditis and septic and pyæmic processes if the cardiac symptoms are not well pronounced. When the fever in malignant endocarditis is distinctly intermittent and continues so for weeks, it might be mistaken for malarial fever unless a sufficient number of blood examinations have been made to exclude malarial infection. It is to be remembered that in some cases of abscess of the liver the symptoms simulate very closely those of malignant endocarditis. The signs and symptoms in such an abscess, however, if carefully investigated, are so distinctive that a correct diagnosis will be reached.

Prognosis.—Malignant endocarditis always terminates fatally; at least I have never seen a recovery when I was perfectly clear in my diagnosis. I have known instances of recovery in recurring endocarditis occurring in debilitated subjects with well marked typhoid symptoms, but the unequivocal signs of malignant endocarditis were not present.

Treatment.—No plan of treatment which has thus far been proposed has seemed to influence the course of this affection. Stimulants should be used freely, and opium in sufficient quantities to relieve the depression which is so marked a feature in its early period. Cold applications to the præcordial region have seemed to me to add to the discomfort of the patient rather than to afford relief.

Chronic or Fibroid Endocarditis.

This form of endocarditis is essentially a fibrosis, and is the foundation of most chronic valvular diseases.

Etiology.—In the majority of instances this affection is secondary to acute rheumatic endocarditis, especially in young subjects. A large percentage of cases, however, have no acute or rheumatic history, and develop so slowly and insidiously as not to be recognized until extensive

fibrotic changes have taken place in the valves or at the valvular orifices. These latter cases are usually associated with general arterial fibrosis, and more or less extensive fibrotic changes will be found in other organs. Alcohol, gout, syphilis, and chronic nephritis are the prominent etiological factors in the production of these changes. Valvular tension from prolonged muscular exertion is claimed to be an important factor in the development of valvular fibrosis, but I question very much the existence of such etiological relations unless high arterial tension exists at the time the prolonged muscular exertion is made. There is a popular belief that valvular disease of the heart is an hereditary affection, but if such cases are carefully analyzed, it will be found that an hereditary fibroid diathesis is at the foundation of the hereditary valvular changes. Chronic endocarditis affecting the right heart is either of fœtal origin or is secondary to mitral insufficiency, and is confined to the tricuspid valve. Chronic endocarditis affects mainly the mitral valve in early adult life, for it is during this period that rheumatism and acute infectious diseases are most common. The aortic valve is its usual seat in persons past middle life.

Pathological Anatomy.—The anatomical changes in this form of endocarditis are sometimes confined to the edges of the valves, at others to their base, or the entire valves may be involved. Any portion of the endocardium of the cardiac cavities may also undergo change similar to that which occurs in the valves: that portion at the apex of the left ventricle is the most frequent seat of such change. The affected portion of the endocardium is either uniformly thickened or fibrous nodules are formed. The essential anatomical change is the production of dense fibrous masses. The lesion which leads to the formation of these masses consists in infiltration of the subendothelial tissue with round cells, which subsequently develop into fibrous tissue. In the aortic valve the tissue about the corpora Arantii is first affected. Later, this fibrous tissue contracts and produces deformity of the valves, or neighboring valvular leaflets may become agglutinated to each other. When the aortic valve is involved, in addition to these valvular changes a dense fibrous ring is formed at the base of the cusps. When the mitral cusps are involved in addition to the thickening, contraction, and adhesion of the edges of the cusps, the chordæ tendineæ become thickened and contracted, drawing the edge of the cusps toward the papillary muscle. These finally become involved, and by their contraction draw the valve into the ventricle, thus preventing it from closing the auriculoventricular opening during the ventricular systole. At the same time, a dense fibrous ring, as in the aortic valve, may form at the base of these cusps. As a consequence, the rigid cusps, whose edges are round and hard, are drawn toward their base, and are thus made to assume a puckered appearance, while they are diminished in length. In this manner the cusps are not only diminished in depth, but not infrequently have their free edges approximated to the cardiac walls, so that extensive valvular insufficiency is the result. This, however, does not always occur, for a thickened valve may have such an abundant fibrinous deposit upon it that the current is obstructed and extensive stenosis results. As the thickening and rigidity of the flaps of a valve increase, their mobility is diminished, and adhesions which begin at their bases

and progress toward their apices take place between their edges. So thoroughly do they become adherent in some cases that all evidence of valve is lost except a fibrous diaphragm across the valvular orifice, having a small slit in the centre which looks and feels like a buttonhole; hence the term "buttonhole slit." Ordinarily the mitral opening will admit the ends of three fingers, but it may become so narrowed that the end of the little finger will scarcely pass through it, and the aortic opening may become so small as not to admit a small quill. These retractions and adhesions cause the mitral valve to assume the form of an inverted cone with an opening at its apex.

Insufficiency and stenosis are often found at the same valvular orifice as the result of the thickening, adhesion, and retraction of the fibrotic tissue. The tendency of the fibrotic tissue which results from this form of endocarditis is to undergo necrotic and calcareous changes. Lime salts are deposited, and finally the entire valve becomes a dense calcareous mass. In connection with these calcareous changes ulceration may be established which will lead to extensive destruction of the valves. The aortic orifice is most frequently the seat of these calcareous changes. The ulcerated and calcareous valves may become covered with fibrinous deposits. It is important to distinguish between the ulcers of malignant endocarditis and the calcareous ulcers of chronic endocarditis. This variety of chronic endocarditis is always dangerous, and when it has once commenced its tendency is to progress. Undoubtedly, it is possible for these fibrotic changes to be arrested at any time during the early stage of the process and the valves to undergo no further changes, but the danger in such cases is that, either from a recurring endocarditis or from other causes, the fibrotic tissue will undergo calcareous changes with ulceration. When chronic endocarditis affects the endocardium of the ventricle the changes will be secondary to and similar to those of valvular endocarditis. In rare instances it may develop independently of valvular diseases. The process is in reality a fibrous overgrowth, and is usually indicated by dense white spots slightly elevated above the surface.

The secondary lesions which take place in the walls and cavities of the heart as the result of chronic endocarditis are a part of the history of chronic valvular diseases, and will be considered in detail under that head.

Chronic Valvular Diseases.

Aortic Stenosis.—Aortic stenosis is a diseased condition of the aortic valve which causes more or less narrowing of the aortic orifice and hypertrophy of the walls of the left ventricle. Almost every case of morbid stenosis is accompanied by some insufficiency.

Etiology.—Typical aortic stenosis is a disease of advanced life, and is always associated with general arterial fibrosis. Rheumatic endocarditis is the most frequent cause of that form of stenosis which is due to valvular changes which not only cause obstruction at the aortic orifice, but allow of more or less regurgitation. Such narrowing may be the result of any of the valvular changes which have been described as taking place in the course of acute or chronic endocarditis. Not infrequently the conditions which give rise to acute or chronic endocarditis

may lead to lesions at the aortic orifice which will obstruct the outgoing blood current, without there having been any evidence of acute endocardial disease. Increased tension of the aorta and prolonged muscular exertion may be the exciting cause of fibrotic changes in the vessel and at the aortic orifice in persons of a gouty diathesis. The frequent development of aortic stenosis in females of a gouty heredity, and who are approaching the menopause, suggests such causal relations.

It is to be remembered that aortic valvular disease is more frequently of non-rheumatic origin than mitral disease.

Pathological Anatomy.—Fibrotic changes in the aortic cusps may render them so rigid that they cannot be pressed back against the walls of the aorta, and their unyielding prominence obstructs the outgoing current of blood. If they become the seat of irregular warty excrescences, as often happens, they still more seriously interfere with the blood current. In rare instances the aortic orifice may be diminished by fibrotic constriction at the base of the valves.

Adhesions of the segments of the aortic valves usually begin at their base and extend along their borders. Sometimes they become fused together into a mass, so that they project into the blood stream in the form of an irregular funnel studded with calcareous nodules; at other times adhesions may take place between the valves with little or no increase of tissue, so that they present the appearance of a thin membrane stretching across the orifice. The most extensive rigidity and calcification of the valves will be found in old subjects, and in such cases the line of attachment of the valves to the aorta becomes entirely obliterated. Obstruction at the aortic orifice is frequently accompanied by atheromatous changes in the aorta, which becomes dilated immediately beyond the aortic ring. In some instances the contraction of the valves at their points of attachment causes them to form deep pockets or pouches behind the cusps. Sometimes the valves are studded with vegetation. As an immediate result of aortic stenosis the wall of the left ventricle becomes hypertrophied. This change is usually a gradual one, and is due to the increased force required to propel the blood through the constricted orifice. If the hypertrophy fully overcomes the obstruction to the outgoing blood current, there will be no change in the size of the cardiac cavities; but if the compensation is incomplete, the cavities will become dilated, the lungs congested, and the right ventricle hypertrophied. A late result is insufficiency of the mitral valves. The amount of blood which is propelled into the aorta at each ventricular systole in a well marked case of aortic stenosis is below the normal, and consequently the supply of blood in the arterial system is diminished and the general nutrition suffers.

Symptoms.—So long as compensation is complete there are no subjective symptoms in aortic stenosis. Even quite extensive stenosis is not incompatible with a condition of perfect health, for a perfect compensatory hypertrophy enables the heart to fill the arterial system and relieve pulmonary pressure. When the ventricular hypertrophy no longer compensates for the obstruction, the arteries are filled inadequately at each systole, the left auricle cannot empty itself freely into the left ventricle, and the pulmonary vessels, as well as the entire venous system, become abnormally full. The diminished arterial supply gives that

pallor to the face which so frequently accompanies this condition, and syncope is liable to occur as a result of cerebral anæmia. These are late effects, and do not usually appear until a condition of more or less mitral insufficiency is reached. As a rule, signs of arterial anæmia, such as pallor, cold hands and feet, loss of muscular power, sense of languor on slight exertion, impaired nutrition, giddiness, nausea, and all the other signs of central anæmia, precede the evidence of venous engorgement. With the venous congestion there are the usual symptoms indicative of engorgement of the pulmonary and systemic circulations.

The pulse in aortic stenosis is normal in frequency, but diminished in volume and force. Usually it is regular in rhythm, although it may be intermittent and jerky in character. The sphygmographic tracings show a slanting or oblique up stroke and considerable separation between the percussion and the tidal waves.

Cerebral embolism is associated with aortic stenosis more frequently than with any other valvular lesions. The embolus usually lodges in the left middle cerebral artery. Sometimes embolism is due to small auricular or ventricular clots that form behind the obstruction. Such clots may occlude the aortic orifice and cause sudden death.[1]

Physical Signs.—Generally the physical signs of aortic stenosis are distinctive and easily appreciated.

Inspection.—In most cases the visible area of the cardiac impulse is abnormally increased, and the apex is displaced downward and outward, frequently accompanied by a lifting of the chest wall over the heart. In old subjects with rigid chest walls and emphysematous lungs, even when there is extensive hypertrophy, there may be no visible impulse.

Palpation.—If the chest walls are rigid, the apex beat may be imperceptible; if they are yielding, it is usually heaving and forcible in character. A systolic thrill of great intensity is often felt at the base of the heart, having its point of maximum force in the second right intercostal space.

Percussion.—The increase in the area of cardiac dulness will correspond to the displacement of the apex to the left, and measures the degree of hypertrophy of the left ventricle.

Auscultation.—Aortic stenosis produces a systolic murmur which more frequently accompanies than replaces the first sound of the heart. The maximum intensity of this murmur is usually at the second sternocostal articulation of the right side. It may be heard, however, with equal intensity over the whole of the upper portion of the sternum, and may be transmitted up the aorta and into the carotids. It sometimes has its maximum intensity at the junction of the third left rib with the sternum. In most cases the first sound of the heart is heard with the murmur, but the murmur may entirely replace or obscure it. This murmur is loud and harsh in character, and is loudest at the beginning of the systole. In pure aortic stenosis the aortic second sound may be inaudible, owing to the thickening and rigidity of the aortic valves, but the pulmonic second sound is always very distinct. The area of diffusion of this murmur follows the law that a murmur is propagated in the direction of the blood current. Sometimes it may be heard in the thoracic and abdominal aorta. It is to be remembered, however, that a

[1] *Pathological Society Transactions*, vol. ix. p. 9.

systolic murmur, audible at the base and carried along the ascending arch toward the sternal end of the right clavicle, is not limited to cases of aortic stenosis, although aortic stenosis always produces a murmur with these characteristics.

Diagnosis.—A rough systolic murmur, with its maximum intensity at the second right costal articulation, transmitted along the ascending arch to the carotids, accompanied by a thrill, a hard, slow pulse, and the evidence of left cardiac hypertrophy in one past middle life, as a rule establishes the diagnosis of aortic stenosis. If one, however, rests exclusively on these signs, he is liable to mistakes, for a roughened and calcified aortic valve with an atheromatous aorta in one who has cardiac hypertrophy due to chronic nephritis may present almost the same signs. It is to be remembered also that systolic murmurs heard in this region may be produced by mitral and tricuspid insufficiency and by anæmia, but the point of the maximum intensity of the murmur and the direction of its transmission will readily distinguish these murmurs from that of aortic obstruction.

The murmur of aortic obstruction is heard with its maximum intensity at the second right sterno-costal articulation, and diminishes in intensity toward the apex. The murmur of mitral insufficiency is heard with greatest intensity at the apex. The murmur of aortic obstruction is transmitted into the vessels of the neck; that of mitral regurgitation to the left, toward the apex beat, and is heard behind, between the fifth and eighth dorsal vertebræ, to the left of the spine, with almost the same intensity as at the apex.

Tricuspid insufficiency also is accompanied by a systolic murmur, but it is rarely audible above the third rib, and its area of diffusion is rarely more than two inches from the point of its maximum intensity at the junction of the left fourth rib with the sternum.

An anæmic murmur is systolic, and heard over the upper portion of the sternum and transmitted into the vessels of the neck, but its maximum intensity is over the carotids. It is soft and blowing in character, and is accompanied by the subjective signs of anæmia and the venous hum.

Thoracic aneurysm may produce murmurs resembling those of aortic stenosis, but the dilating impulse, the single or double bruit, the pain, and the usual symptoms of thoracic aneurysm are absent when aortic stenosis alone is present. Besides, aneurysmal murmurs have their maximum intensity at the seat of the aneurysm, and not at the base of the heart.

Aortic Insufficiency.

Aortic insufficiency is produced either by an abnormal condition of the aortic valves or by dilatation of the aortic orifice, which prevents its complete closure during the cardiac diastole and allows a backward current of blood to flow from the aorta into the left ventricle.

Etiology.—It is most frequently met with in vigorous, athletic men about the middle period of life. Acute endocarditis is rarely a cause of aortic insufficiency, unless it is attended by extensive ulcerations, when it may lead to a rapidly fatal termination. Its most frequent cause is fibrotic endocarditis, which leads to thickening, induration, and

contraction of the valves. Such fibrotic changes are so often met with in persons who have been subjected to violent and prolonged physical exertion, who give no history of rheumatism, that persistent tension of the valves during the cardiac systole must be regarded as a prominent etiological factor in its development. Acting in a similar manner, alcohol becomes a very potent factor, especially if it is added to the tension of muscular strain. Syphilis is a not infrequent cause of fibrotic changes at the aortic orifice which give rise to aortic insufficiency. Although rheumatism has been regarded as a prominent causal factor in its production, one fails to elicit a rheumatic history in the majority of those who give evidence of aortic insufficiency, and in this respect it differs from mitral insufficiency, which in the majority of cases is the result of rheumatic endocarditis.

Congenital insufficiency is exceedingly rare, for although two segments of the valve may be united, the valve is rarely rendered insufficient from such a cause.

Pathological Anatomy.—In aortic insufficiency the cusps are prevented from performing their normal function on account of the following anatomical changes: As a result of chronic endocarditis the cusps may have been thickened, puckered, and shortened, so that they do not meet when brought into the plane of the orifice. When the central portion of the segment is indurated, the whole valve subsequently curls up, either toward the orifice or back against the wall of the aorta, and in either case there is insufficiency of the cusps. In the one case there is insufficiency with great obstruction; in the other, with but very slight obstruction. These processes of thickening and shortening may also result from fibrotic changes extending from the aorta to the cusps. Regurgitation may result also from adhesion of the valve tips to the walls of the aorta or the rupture of a segment of a valve. These fibrotic changes may be combined with atheroma and calcification. Insufficiency from dilatation of the aortic orifice, the result of extensive fibrotic changes in the ascending portion of the aorta, is not infrequent.

During diastole, normally, the blood is passing from the auricle into an emptied ventricle; when however, regurgitation has persisted for a considerable time, there will be added to the primary stream, which of itself is capable of filling the cavity of the ventricle, a regurgitated stream from the aorta; and by this combination of two streams the left ventricle becomes over-distended and permanently dilated. This dilatation occurs all the more readily since during the diastole the ventricular walls are relaxed and less capable of resisting the increased blood pressure. This permanent dilatation of the left ventricle occurs in a comparatively short time, and to overcome the dilatation and the obstruction to the cardiac circulation, which allows of over-distention of the ventricular cavity, the walls of the ventricle hypertrophy. The hypertrophy goes on increasing until it compensates for the dilatation, but before this point is reached the ventricular cavity sometimes becomes very much dilated and the left heart reaches an enormous size. The dilatation, and in such cases often hypertrophy, reach their extreme limit, the heart weighing thirty or forty ounces; Fagge records a case in which its weight was forty-eight ounces. This is sometimes called the "bovine heart." In such a case the heart has a peculiar pointed form, the right

ventricle appearing like a mere appendix. The left ventricle becomes capable of containing an abnormally large quantity of blood, so that the arteries are over-distended with each systole. The concussion caused by the increased ventricular power forcing an abnormally large quantity of blood into the aorta causes extensive fibrotic changes in its walls, and results in its dilatation.

The coronary arteries become secondarily involved in the fibrotic changes, their orifices are narrowed, and the nutrition of the hypertrophied cardiac walls is consequently diminished. In the normal heart the aortic recoil is undoubtedly the principal force which propels the blood into the coronary arteries. When the aortic valves are insufficient and furnish little or no resistance to the return blood current, we have another factor in diminishing the coronary blood supply; therefore in all cases of aortic insufficiency with extensive dilatation and hypertrophy of the heart, degenerating changes must sooner or later take place in the cardiac muscle.

In some cases atrophy of the papillary muscles allows the mitral flaps to swing back into the left auricle when increased pressure is exerted upon them; when from any cause mitral incompetency becomes secondary to and coexistent with aortic insufficiency, all the signs of impeded venous circulation will be present. When over-distention of the left ventricle causes incomplete emptying of the left auricle, a greater or less amount of passive hyperæmia of the lungs may be present without mitral insufficiency. The arteries of the body undergo fibrotic change as the result of the increased quantity of blood which is forced into them with each forcible ventricular systole.

Symptoms.—So long as aortic insufficiency is fully compensated for by hypertrophy of the left ventricular walls, the individual will suffer little or no inconvenience, even with considerable insufficiency. Uncomplicated aortic insufficiency often exists for years without giving rise to any sensations of distress about the heart that will attract the attention of the patient or in any way interfering with a moderately active life. In those cases in which the aortic insufficiency is associated with or is a part of a general arterio-sclerosis the failure in compensation occurs earlier than in those in whom the arteries are comparatively normal. As soon as there is the slightest failure in compensation the heart action becomes excessive during excitement or violent muscular effort, which causes the individual to become anxious and irritable, and he is generally aware that exercise will augment his uncomfortable symptoms. As the hypertrophy increases they suffer from dyspnœa, cardiac palpitation, vertigo, flashes of light, a feeling of faintness on rising suddenly, and they are forced to sleep with their heads elevated. On slight exertion the patient experiences a sense of cardiac oppression and palpitation. There is a visible pulsation in the arteries of the head, neck, and upper extremities. Pain in the præcordial region, in the left shoulder, or extending down the left arm becomes a troublesome symptom, and is usually of a distinctly shooting or stabbing character. Sometimes this pain is accompanied by numbness and a peculiar whiteness of the skin along the line of pain. In certain cases the pain shoots from the middle of the sternum to the right arm. As the failure in compensation increases attacks of dyspnœa occur at night,

and the patient is often unable to lie down on account of the difficulty in breathing which comes on as soon as he assumes the recumbent posture. The feet become œdematous, and in some instances the œdema gradually extends upward until a condition of general anasarca is reached. The face becomes puffy and cyanotic.

In the advanced stages of aortic insufficiency there is orthopnœa and angina pectoris. Attacks of syncope at first occur only on exertion, but later they come on independently of it, and are very distressing.

The pulse of aortic insufficiency is characteristic. It was first described by Sir Dominick Corrigan, and is usually called the "Corrigan pulse." The pulse is sometimes spoken of also as the "piston pulse." It is large and distinct, quickly projected against the finger, and just as quickly the arterial tension sinks and the impulse vanishes. Sometimes it is accompanied by a vibrating jar. Its characteristics are more apparent when the arm is raised above the head. Although quick and jerking, it is always regular in rhythm. The radial pulse is felt a little after the apex beat; thus the pulse wave of aortic regurgitation travels slowly along the arteries. The delay in the pulse is always constant. Pulsation of the retinal arteries can often be detected by the ophthalmoscope.

In advanced aortic incompetency patients become irritable and peevish. Sometimes delirium with hallucinations and suicidal tendencies develop. I have occasionally found it necessary to restrain these patients to prevent them from injuring themselves or their attendants.

Physical Signs.—Inspection shows a forcible and increased area of cardiac impulse, sometimes reaching to the seventh interspace and laterally to the left axillary line. The vessels of the neck are seen to pulsate forcibly, and at times all the superficial vessels have a distinct throbbing impulse. The so-called "capillary pulse," although sometimes seen in aortic incompetency, does not have any constant or necessary connection with it.

Palpation.—A heaving, lifting impulse will usually be perceived over the præcordial region which may be transmitted over a large portion of the anterior chest wall. When there is extensive dilatation of the left ventricle the impulse will become diffused and indistinct. Occasionally a continuous diastolic thrill, equally intense during the whole of the diastole, will be felt over the sternum, most distinctly at the site of the aortic valves.

Percussion.—The area of percussion dulness corresponds to the degree of cardiac enlargement. Deep dulness is elicited below and to the left of the normal cardiac area, and its outline is more oval than in health. So soon as dilatation exceeds the hypertrophy the area of dulness will extend laterally rather than vertically. The area of dulness may extend six to eight inches from right to left and from the upper edge of the third rib to the line of the liver dulness. The superficial area of dulness may be increased laterally and toward the left.

Auscultation.—Aortic incompetency is characterized by a diastolic murmur which may take the place of or immediately follow the second sound of the heart. It is distinct at any point over the base of the heart, but usually has its maximum intensity either at the sternal end of the second right costal cartilage in the second right intercostal

space, or at the sternal junction of the third rib on the left side. It is transmitted over the sternum, and sometimes will be loudest at the xiphoid cartilage. Sometimes it is transmitted toward the apex. Its area of diffusion is greater than that of any other murmur. It is not only conducted down the sternum to the xiphoid cartilage and to the apex, but sometimes it may be heard faintly in the transverse and descending portions of the arch, along the spinal column, and even in the radial and femoral arteries. Foster states that incompetency of the posterior segment of the valve produces a murmur which is conducted to the apex, whereas inadequacy of either or both of the anterior segments is accompanied by a murmur which is conducted to the ensiform cartilage. This point has a practical bearing on account of the relationship of the anterior segments of the valves to the coronary artery. If the murmur indicates a lesion of the posterior flap of the valve, the prognosis is better. The murmur of aortic incompetency may be soft, blowing, or rough, and is frequently musical in character. It is loudest at the beginning of diastole and gradually decreases in intensity. If aortic stenosis coexists with aortic incompetency, there will be a double murmur, having its maximum intensity at the right side of the sternum in the second intercostal space. The systolic and diastolic portions of such murmurs are sometimes separated from each other by a distinct pause.

If mitral incompetency is associated with aortic incompetency, each murmur will retain its own area of maximum intensity and diffusion. Murmurs of aortic incompetency are sometimes very indistinct, and can only be heard when the patient is in the recumbent posture. The loudness of the murmur is not a measure of the extent of the incompetency. A diastolic murmur heard at or below the line of the aortic valves, chiefly audible in the centre of the sternum, indicates extensive aortic incompetency. If a diastolic murmur is audible in the carotids, it is invariably preceded by a loud systolic murmur.

Mitral Stenosis.

Usually stenosis and insufficiency of the mitral orifice occur together, and stenosis probably never occurs without some insufficiency.

Etiology.—Mitral stenosis is almost always the result of valvular endocarditis occurring in children or young adults. It is rarely met with after the fifteenth year. It is nearly twice as frequent in females as in males. A limited number of cases seem to be congenital. Acute rheumatic endocarditis is its most frequent cause. I have never been able to trace a typical case of stenosis to any other cause.

Pathological Anatomy.—The morbid changes which give rise to mitral stenosis are—thickening and contraction of the valve segments, adhesion of the free edges of the valve tips, and agglutination of the chordæ tendineæ. The free edges of the valves are often greatly thickened, and calcareous masses may develop in the thickened portion. When the chordæ tendineæ and papillary muscles have become adherent the edges of the valves are drawn down into the cavity of the heart. As the flaps are adherent along their adjacent borders, the valve presents a funnel-shaped appearance with its base looking toward the auri-

cle, and with a slit-like opening into the ventricle below which may scarcely admit the tip of the little finger. This has been called the "buttonhole" slit. Sometimes the flaps are stretched horizontally across the auriculo-ventricular ring like a diaphragm, with a small crescentic opening in the centre. In rare instances the valve segments are normal, and the stenosis results from large calcareous masses which project into the mitral opening from the auriculo-ventricular ring. As a result of mitral stenosis the left ventricle becomes smaller, and sometimes its walls become thinner than normal. The aorta also is smaller and the walls are thin. There are always dilatation and hypertrophy of the left auricle. Sometimes the auricle is enormously dilated, so much so that Thurman fifty years ago described it as a true aneurysm of the left auricle. Not infrequently the auricular walls measure an eighth of an inch in thickness. Its appendix is elongated, assuming a peculiarly curved form, the aperture between which and the auricle proper becoming larger than normal. Sometimes it reaches two and three quarter inches in length. For a time the auricular hypertrophy overcomes the obstruction to the blood current at the auriculo-ventricular opening, but gradually the compensation is broken and the pulmonary circulation becomes obstructed, causing congestion of the pulmonary veins. To overcome this the right ventricle hypertrophies, and for a time is able to counteract the pressure in the pulmonary vessels, but sooner or later dilatation occurs with incompetency at the tricuspid orifice, and congestion of the systemic veins follows. The pulmonary congestion which results from the obstruction in the pulmonary veins may lead to those changes which collectively constitute brown induration of the lung and thrombosis of the right ventricle.

Symptoms.—Mitral stenosis causes few subjective symptoms. Active physical exertion induces cardiac palpitation, but this will cease as soon as the auricle can relieve itself, which is readily accomplished by the patient's lying on the right side with the head slightly elevated. These patients are usually pale and anæmic. Anginal pains are frequently felt in the region of the apex. As long as compensation is maintained by the auricular hypertrophy the pulse is regular and normal in character. When the left ventricle no longer receives and discharges the normal quantity of blood, the pulse becomes small, feeble, rapid, and irregular. The pulmonary congestion which attends the advanced stage of mitral stenosis causes continual dyspnœa, which is increased by physical exertion and is accompanied by a dry hacking cough. After prolonged or violent physical exertion a large quantity of glairy, watery mucus may be expectorated in a few moments or a profuse watery, blood-stained expectoration indicative of pulmonary congestion and œdema may occur. I have known the exertion of walking rapidly against a strong wind to induce such intense congestion and œdema of the lungs in one with extensive mitral stenosis as to cause sudden death.

Orthopnœa is not a frequent symptom of mitral stenosis, for even in extensive and long-standing cases the pulmonary circulation is maintained, since the auricle is usually able to empty itself, and only becomes engorged during active physical exertion. It should be mentioned here that the old idea, that pure mitral stenosis produces hypertrophy of the left ventricle, is fallacious.

PHYSICAL SIGNS.—*Inspection.*—The cardiac impulse is usually feeble and indistinct. Sometimes it has a visible undulating movement. It is rarely perceptible to the left of its normal area, but is usually most distinct in the fourth interspace near the sternum.

Palpation.—The apex beat is less forcible than normal. A distinct presystolic, purring thrill will be communicated to the hand. This thrill is the diagnostic sign of mitral stenosis. It is most distinct in the region of the apex. It begins during the diastole, and increases in intensity up to the commencement of the ventricular systole. It is often perceptible as high as the third interspace, and sometimes as high as the second. It may be diffused over the whole præcordial area and may continue throughout the entire cardiac systole.

Percussion.—The area of cardiac dulness will be increased to the right and upward. If the left auricle is greatly dilated, the increase in the dulness will be upward along the left border of the sternum.

Auscultation.—Mitral stenosis is characterized by a loud churning, blubbering presystolic murmur. This murmur is longer in duration than any other murmur. It ends at the commencement of the first sound, being synchronous with the purring thrill. It is heard with its maximum intensity a little above the apex beat. It is always louder when the patient is erect than when in the recumbent posture. It is rarely conveyed to the left of the apex beat, and loses in intensity as the stethoscope passes to the right of the sternum or above the third rib. The second sound of the heart in the second left interspace is loudly accentuated and sometimes reduplicated. If the murmur immediately follows the second sound and continues through to the commencement of the first, it indicates a diaphragmatic construction of the mitral orifice. When mitral stenosis and incompetency coexist the two murmurs run into each other, constituting a single murmur. The harsh character of the presystolic element of the murmur can always be recognized. As compensation fails a stenotic murmur loses its intensity and often becomes inaudible; if the compensation is restored, the murmur reappears. If the patient is seen only during the period of broken compensation, the stenotic disease may pass unrecognized.

MITRAL INCOMPETENCY.

Incompetency at the mitral orifice is due to a condition of the mitral valve which permits the blood to flow back from the left ventricle into the left auricle during the ventricular systole.

ETIOLOGY.—Rheumatic endocarditis is the primary cause of most of the changes which lead to mitral insufficiency. It may occur in cases of extreme anæmia or be due to changes in the muscular substance of the walls of the left ventricle, which markedly diminish its contractile power. It may result from the stretching of the left auriculo-ventricular orifice which accompanies dilatation of the left ventricle, secondary to changes at the aortic orifice. Disease of the columnæ carneæ and chordæ tendineæ, which allows the flaps of the valve to pass beyond the plane of the orifice, will cause mitral incompetency. It may occur at any age, but it is most frequently met with in the young.

PATHOLOGICAL ANATOMY.—The most common lesions which give

rise to mitral incompetency are thickening, induration, and shortening of the flaps from chronic endocarditis. The valve may be rendered incompetent also by contraction of the chordæ tendineæ, by adhesion of the valve leaflets to the ventricular wall, or by their rupture or that of the chordæ tendineæ.

Calcareous plates at the base of the valve may prevent the closure of segments of the valve, or all the structures at the mitral orifice may be converted into a firm calcareous mass. In such cases more or less obstruction accompanies the regurgitation.

Mitral insufficiency due to such great dilatation of the ventricle as to render it impossible for the mitral segments to close the auriculo-ventricular orifice is rarely met with except with aortic insufficiency.

Mitral incompetency causes dilatation of the left auricle, due to the pressure of two blood currents during its diastole, one from the lungs and the other from the left ventricle. This dilatation is followed by thickening and hypertrophy of the auricle. Following this, the pulmonary circulation is impeded, the pulmonary vessels become dilated, and their walls undergo fibroid changes as the result of the continued regurgitant pressure, and the lung tissue undergoes those changes which are incident to passive congestion of the pulmonary vessels. This constant obstruction to the return circulation of the lungs interferes more or less with the outward current of blood to the lungs from the right ventricle. As the obstruction comes on gradually, the right ventricle becomes hypertrophied, so as to overcome it. Consequently, the hypertrophy of the right ventricle compensates at first for the mitral insufficiency, and as long as the right ventricle is able to overcome the increased resistance in the lungs the systemic circulation is not impaired. But after a time this compensatory hypertrophy ceases, the right auricle becomes dilated and hypertrophied, the tricuspid valve becomes incompetent, and congestion of the systemic veins results. This impedes the hepatic and portal circulations, and is indicated by passive congestion of the abdominal viscera and by cyanosis of the surface during active physical exercise. Not infrequently moderate mitral incompetency with considerable hypertrophy and dilatation of the left auricle and ventricle may exist for years without any disturbance of the pulmonary or systemic circulation, but there is constant danger in such cases that sudden overtaxing of the heart will break the compensation and give rise to alarming symptoms.

Acute dilatation of the left ventricle, the result of disease at the aortic orifice, may cause a relative incompetency of the mitral valve, which is sometimes immediately followed by extensive dilatation of the left auricle and intense engorgement of the lungs.

Symptoms.—During the early stage of mitral incompetency, unless the incompetency comes on suddenly from rupture of the valve or of the chordæ tendineæ, there are no subjective symptoms; and so long as hypertrophy of the right ventricle perfectly compensates for the insufficiency, even though the hypertrophy is extreme, the patient will not be made aware of its existence. But when the right ventricle fails to overcome the obstruction in the pulmonary circulation caused by the regurgitant blood current, and the stage of commencing failure of com-

pensation is reached, there will be dyspnœa on exertion, accompanied by cyanosis and a hacking cough, with expectoration of frothy serum. Sometimes the serum is blood-stained and there may be attacks of quite free hæmoptysis, although it should be remembered that profuse hæmoptysis is far more frequent with mitral stenosis than with incompetency. Cardiac palpitation is a frequent attendant of this stage.

In the advanced stage, when the period of broken compensation is reached, the lips, face, and finger-ends become blue and the symptoms of extensive venous engorgement are present. The heart action becomes weak and irregular, dyspnœa and cough are constant, and the watery, bloody expectoration contains brown pigment granules. The liver is enlarged, and the patient will complain of a sense of weight and fulness in the hypochondrium, and there will be anorexia, nausea, and a sense of oppression in the epigastrium. Sometimes the hepatic circulation becomes so obstructed that jaundice will be added to the cyanotic discoloration, which gives to the surface a peculiar greenish hue. Congestion of the portal circulation causes frequent attacks of gastric and intestinal catarrh, and evidences of embarrassed renal circulation are present. The urine is high colored and loaded with urates, and may contain albumin and blood casts. Headache, dizziness, vertigo, stupor, somnolence, and sometimes a peculiar form of delirium often occur. A late symptom of mitral incompetency is dropsy, beginning in the feet and gradually extending to the trunk and serous cavities. It may require several years or only a few months before a condition of general anasarca is reached. With the anasarca the dyspnœa becomes extreme, the patient being unable to lie down for more than a few moments at a time.

In this stage the occurrence of hemorrhagic infarctions in the lung is not infrequent. As these symptoms come on gradually, and as the compensation may be temporarily restored by treatment, these patients are apt to become very hopeful and to expect much from their medical attendant, but, ultimately they reach a condition for which no relief can be given, and they die either from general dropsies or from excessive cardiac dilatation. Death usually takes place slowly.

The pulse of mitral incompetency before the period of failing compensation remains regular in force and rhythm; after the stage of failing compensation has been reached it becomes diminished in force and volume, irregular in rhythm, and increased in frequency, but never jerking in character. When the heart's action is excited it has a certain tremulousness. These last-named characteristics are to be regarded more as the result of the changes in the myocardium than of the valvular insufficiency. If the pulse of mitral insufficiency has any distinctive characteristic, it is its diminution in volume.

Physical Signs.—*Inspection.*—The visible area of cardiac impulse is abnormally increased and is more or less distinct, according to the extent of the right ventricular hypertrophy. Sometimes in children there will be bulging of the præcordium and a heaving of the thoracic wall with each systole, and not infrequently there is an epigastric impulse synchronous with the heart beats. In aggravated cases a double impulse often accompanies the cardiac systole, and is due to a non-coincidence in the contraction of the ventricles. The jugular veins

may be distended and have a wavy impulse when the patient is in a recumbent posture.

Palpation.—The apex beat is displaced to the left. If the hypertrophy predominates over the dilatation, it is felt below the normal area; when the dilatation exceeds the hypertrophy, the apex beat is carried outward and slightly upward. The impulse is diffused and more or less forcible according as the right or left ventricular hypertrophy predominates. A systolic tremor, felt most distinctly at the apex and becoming less intense the farther the hand is removed either to the right or left from that point, is invariably due to mitral insufficiency. I have never, however, met with a distinct purring thrill in mitral incompetency unassociated with mitral stenosis.

Percussion.—The area of superficial as well as deep dulness is increased laterally and downward. There is no valvular disease which produces such extensive lateral increase in the area of dulness as mitral incompetency.

Auscultation.—Mitral incompetency causes a systolic murmur which either completely or partially replaces the first sound of the heart. The quality of the murmur is variable, and not in itself as distinctive as that of mitral stenosis. It is usually soft and blowing in character. Sometimes toward its end the murmur becomes distinctly musical in character. It is heard with its maximum intensity at the apex, and its area of diffusion is to the left. It can be heard at or near the inferior angle of the left scapula, and is usually as distinct between the lower border of the fifth and the upper border of the eighth vertebra at the left of the spine as at the apex. It varies in intensity with the position of the patient. It may be present in the recumbent and absent in the erect posture. Accentuation of the pulmonary second sound is an important sign of mitral incompetency, and is heard with maximum intensity in the second interspace to the left of the sternum. Skoda first drew attention to this sign, and regarded it as an infallible indication of mitral regurgitation.

When mitral incompetency and stenosis coexist a continuous murmur is heard, which begins usually after the second heart sound and continues throughout the cardiac cycle. The two murmurs, although mingling to form one, can in most instances be readily distinguished from each other, for the point of maximum intensity and the very limited area of diffusion of a presystolic murmur readily distinguish it from a mitral systolic. It is important to recognize the existence of both these murmurs in estimating the prognosis in any case.

DIAGNOSIS.—The diagnosis of mitral incompetency is easily made. The rhythm and area of diffusion of the murmur which attends it are sufficient to distinguish it from other valvular lesions. The character of the pulse, the symptoms referable to the right heart, and the pulmonary complications will also assist in its diagnosis. Sometimes systolic murmurs are produced in the left ventricle which simulate very closely the murmur of mitral insufficiency, but such murmurs are not associated with ventricular hypertrophy or accentuation of the pulmonary second sound. I do not believe it is possible in all instances to determine by its character whether a murmur of mitral insufficiency is due to a lesion of the valve segment or to relative mitral insufficiency from dilatation

of the mitral ring, but the history of the case, the condition of the arteries, and the presence of aortic valvular insufficiency will assist in the differentiation.

Pulmonary Stenosis.

On account of the infrequency of disease of the pulmonic valves very little can be said of the phenomena to which such disease may give rise. Its diagnosis is arrived at only by exclusion, and it cannot be recognized except by the physical signs which attend it, and even they are often misleading.

Etiology.—As has already been stated, endocarditis in the right heart is rare except in intra-uterine life. The etiological factors of aortic valvular disease have no analogues in the pulmonary vessels. Pulmonary stenosis rarely results from endocarditis or disease in the pulmonary artery. It is almost always a congenital affection. When abnormal communication between the two sides of the heart exists, endocarditis in the right heart may be excited by the arterial blood, but such an occurrence is exceedingly rare. Congenital syphilis has been considered a possible cause of pulmonic stenosis.

Pathological Anatomy.—There is usually adhesion of the valve segments. Bertin records an instance of pulmonic stenosis where the adherent valves formed a horizontal septum across the pulmonic orifice, the opening being barely one fourth of an inch in diameter. A thickened and projecting tricuspid valve has been found to be the cause of obstruction at the pulmonic orifice, the pulmonary valves themselves being normal. Obstruction of the pulmonary artery just beyond the valves may be caused by the pressure of aneurysms, tumors of the anterior mediastinum, and enlarged bronchial glands. Long-standing obstruction at the pulmonary orifice is followed by compensatory hypertrophy of the right ventricle, accompanied by tricuspid regurgitation and dilatation of the right auricle. I have met with three cases where an obstructive pulmonic murmur was heard during life, and at the autopsies tumors were found pressing on the pulmonary artery, diminishing its calibre.

Symptoms.—The subjective symptoms which have been described in the few cases of pulmonic stenosis reported are neither constant nor diagnostic. In some cases murmurs existed, in others there were cardiac palpitation, dyspnœa, cyanosis, and anasarca; none of which belong exclusively to a pulmonic lesion nor do they necessarily depend upon it.

Physical Signs.—*Inspection*, *palpation*, and *percussion* give negative rather than positive results. In a few instances an appreciable thrill was felt, confined to the region of the second left intercostal space near the sternum.

Auscultation.—A systolic murmur may be heard with maximum intensity immediately over the pulmonic valves. It is very superficial, very distinct, and limited in its diffusion. It is not transmitted to the xiphoid cartilage nor along the course of the aorta nor into the vessels of the neck. The pulmonary second sound is weak or absent. It may be obscured by a diastolic murmur. Hypertrophy of the right ventricle may be present.

DIAGNOSIS.—The diagnosis of pulmonary stenosis, if reached at all, is reached only by exclusion. It may be differentiated from aortic stenosis by the absence of the characteristic pulse of the latter, the absence of left ventricular hypertrophy, and the fact that the murmur is not transmitted into the vessels of the neck. It is impossible to distinguish a murmur produced by pulmonic stenosis from one produced by pressure on the pulmonary artery, by an aneurysm at the sinus of Valsalva, or a tumor in the anterior mediastinum, nor can it be distinguished from the soft blowing murmurs sometimes produced by the pressure of tubercular consolidation.

PULMONARY INCOMPETENCY.

This form of valvular lesion is exceedingly rare; indeed, many doubt its occurrence. There are, however, a few well authenticated cases in medical literature the result of injury or congenital malformation. The condition is so very rare and of so little practical significance that it does not require separate consideration.

TRICUSPID STENOSIS.

This valvular defect is usually of congenital origin, and is associated with other congenital malformations which lead to early death. There are a few recorded cases of acquired stenosis associated with mitral stenosis. In some of these cases the tricuspid flaps were thickened and united for one third of their length; in others the valve flaps formed a diaphragm with a central opening scarcely admitting the point of the index finger. In all the reported cases there was cardiac hypertrophy. In a case which recently occurred in my wards in Bellevue Hospital the tricuspid opening would not admit the end of the little finger. It was associated with extensive mitral stenosis and hypertrophy of both auricles and ventricles. The chief subjective symptoms in this case, as in all the recorded cases, were extreme lividity of the face, constant dyspnœa, vertigo, albuminous urine, and in the late stages general anasarca.

PHYSICAL SIGNS.—Inspection shows a jugular pulsation and an epigastric impulse. A venous thrill will usually be felt at the base of the neck. The area of cardiac dulness will be abnormally increased laterally and upward to the right. On auscultation a presystolic murmur is heard, with its maximum intensity at the lower portion of the sternum just above the xiphoid cartilage. This murmur may be transmitted faintly toward the base of the heart, but never toward the apex.

The DIAGNOSIS of this lesion can only be made by exclusion. The murmur of mitral stenosis, with which tricuspid stenosis is almost always associated, is limited to the apex; the murmur of tricuspid stenosis, having the same rhythm, is heard at the base of the ensiform cartilage, and between these points there is an area where no murmur is heard.

The PROGNOSIS and TREATMENT will depend upon the extent and gravity of the accompanying mitral stenosis.

Tricuspid Incompetency.

Etiology.—Regurgitation at the tricuspid orifice may be the result of an endocarditis which has thickened and puckered the valve, but its most frequent cause is stenosis or incompetency of the mitral valve. Any condition of the lungs which will produce dilatation and hypertrophy of the right ventricle may lead to it. It is met with in extensive pulmonary emphysema, in pulmonary fibrosis, and in advanced chronic bronchitis.

Pathological Anatomy.—When it is the result of endocarditis the tricuspid valve may be thickened and retracted or adjacent flaps adherent, and the columnæ carneæ and papillary muscles shortened. Obstruction in the pulmonary circulation, either secondary to mitral disease or to emphysema and fibrosis of the lung, causes right ventricular dilatation, which renders the tricuspid valve relatively insufficient. The first effect of tricuspid insufficiency is dilatation and hypertrophy of the right auricle. When the valves in the subclavian and jugular veins become unable to resist the regurgitant current of blood, jugular pulsation follows. Before this occurs, however, the branches of the inferior vena cava and the organs to which they are distributed become engorged, for they have no valves to resist the backward flow of blood. The inferior cava and the hepatic veins sometimes become greatly distended under these circumstances, and the liver presents the peculiar appearance described as "nutmeg liver." Later, the skin assumes a dingy yellow hue. If cyanosis is present, a peculiar greenish tint is produced which is met with only in tricuspid incompetency. The spleen is enlarged and hard, the mucous membrane of the stomach is congested, and often presents numerous ecchymoses and hemorrhagic erosions. Intestinal catarrh develops, and the general venous congestion in the abdominal cavity causes hemorrhoids and ascites. The kidneys become congested and have a hard, stony feel. Thrombi may form in the femoral veins. The venous stasis in the lower parts of the body is followed by transudation of serum first in the ankles, and then the dropsy progresses upward until finally the condition of general anasarca is reached.

Symptoms.—Tricuspid insufficiency being usually secondary to some mitral lesion or chronic pulmonary affection, its symptoms during its early stage are vague and masked by those of the primary affection. But as soon as the valves become so inefficient that the venous return is markedly impeded, a train of symptoms is developed which have their origin in the visceral changes already referred to. With extensive tricuspid insufficiency there may be cardiac palpitation, dyspnœa, and marked irregularity in the force and rhythm of the heart. The hepatic and splenic areas of dulness are increased, the skin becomes shiny, and there is obstinate constipation. Dyspeptic symptoms are prominent and the urine is scanty, dark colored, and of high specific gravity. Marked cerebral hyperæmia is manifested by headache, dizziness, vertigo. There is a peculiar mental disturbance which is not met with in any other form of heart disease. If the patient is placed in a recumbent position, the face becomes turgid and blue, and if he remains long in such a position, stupor and coma may supervene. A very late symptom

is dropsy, which begins at the ankles and extends upward until a condition of general anasarca is reached, the genital organs rarely becoming œdematous.

PHYSICAL SIGNS.—*Inspection.*—The visible area of the cardiac impulse is increased more in extensive tricuspid insufficiency than in any other valvular lesion. It sometimes extends from the left nipple to the xiphoid cartilage. There is an impulse in the jugular veins, more apparent in the right than in the left. The veins in the face, arms, and hands may pulsate.

Palpation.—The apex beat is diffused and feeble except when the left ventricle is greatly hypertrophied. There is distinct epigastric pulsation, due to reflux into the veins of the liver, which is synchronous with the cardiac impulse.

Percussion will reveal an increase in the area of cardiac dulness upward and to the right, sometimes as high as the second intercostal space.

Auscultation.—The murmur of tricuspid insufficiency occurs with, or takes the place of, the first sound of the heart. It is superficial, low pitched, faint, soft, and blowing, and is heard with greatest intensity over the lower part of the sternum. As a rule, it is inaudible above the third rib or to the left of the apex beat. Sometimes it is transmitted from the base of the xiphoid cartilage two or three inches upward to the right of the sternum. When it is audible only over a limited area it may be overlooked.

DIAGNOSIS.—The murmur of tricuspid insufficiency may be confounded with the murmurs of aortic and pulmonic obstruction. It is to be remembered, however, that a tricuspid regurgitant murmur is never audible above the third rib, is not attended by accentuation of the pulmonic second sound, and is accompanied by jugular and epigastric pulsation. Its point of maximum intensity is near the base of the ensiform cartilage. It is not difficult to differentiate it from a mitral regurgitant murmur if the rules for the diagnosis of such a murmur are observed.

PROGNOSIS IN VALVULAR DISEASES.

So long as full compensation is maintained in any form of valvular disease, although the lesion be a serious one, the immediate prognosis is good. Any general statements as to duration of life in valvular diseases and their relative frequency as causes of death are unreliable. Each case must be judged separately. There are certain conditions other than the valvular lesion which are more important guides in our prognosis than the lesions themselves. Age is an important factor in the prognosis of any case of valvular disease. The outlook in very young children is always unfavorable, for the reason that the valve lesion is apt to be progressive on account of the liability to recurrence of the rheumatic attacks. Valvular lesions which develop in early adult life are more likely to be permanently compensated than those which develop in middle life or in children. The prognosis in valvular diseases which develop in middle life in those who give evidence of extensive arterial changes is always unfavorable. In old age extensive

valvular insufficiencies are well borne and give rise to a few urgent cardiac symptoms.

Women bear valvular lesions better than men, except during the childbearing period, for arterial changes are less likely to occur in them and they lead quieter lives. Occupations, habits of life, and climate all necessarily influence the prognosis in valvular diseases. If one with valvular insufficiency is compelled to perform active physical labor, and is exposed to the vicissitudes of a cold, damp climate, the liability to early failure of compensation is very great. The same is true if one is indiscreet in diet or is addicted to the excessive use of alcohol or tobacco or indulges in venereal excesses. Intercurrent diseases of all kinds render the prognosis grave.

The order of relative gravity in the different valvular lesions may be estimated in a general way as follows: First in order of gravity stands tricuspid incompetency; second, aortic incompetency; third, mitral stenosis and incompetency; fourth, aortic stenosis; and fifth, pulmonic stenosis.

Tricuspid incompetency is so constantly secondary to diseases of the other valves that its occurrence becomes a part of the history of the valvular lesion which it complicates. When, however, it becomes extensive, the duration of life is limited to a short period, as it has no possible compensation and leads rapidly to such obstruction in the general systemic circulation that the life of the patient is terminated by cyanosis and general anasarca.

Aortic incompetency must always be regarded as a grave form of valvular disease. It is impossible to estimate the probable duration of life with any approach to certainty, for the coronary arteries may not for a long time become sufficiently involved to render the case a serious one. Sooner or later, however, they will undergo changes which will cause the outlook to become more serious. It must always be borne in mind that when fibrotic changes take place at the origin of the aorta the coronary arteries are liable to undergo similar changes, and under such conditions sudden death is possible from embolism of one of their branches. If the valvular lesion is of rheumatic origin, aortitis and obstructive changes in the coronary arteries are of rare occurrence; consequently the prognosis in such aortic insufficiencies is comparatively good, except in young children. When in aortic insufficiency cyanosis and dropsy result from the failure of a dilated and hypertrophied left ventricle to empty itself, the prognosis is unfavorable. Acute ventricular dilatation produced during violent or prolonged physical exertion in one who has extensive aortic insufficiency is rarely recovered from, and marks the commencement of a period which soon terminates in fatal heart insufficiency. When the hypertrophy and dilatation are moderate in degree, and there are no urgent cardiac symptoms and no signs of extensive arterial changes, aortic incompetency may be fully compensated for years; but if it is complicated by mitral disease, with a dilated feeble ventricle and extensive arterial changes, the prognosis becomes exceedingly unfavorable.

The prognosis in aortic stenosis is less grave than in any other valvular lesion. Life may be prolonged and good health enjoyed for many years if the ventricular hypertrophy fully compensates for the obstruction; but as soon as the compensatory hypertrophy fails to overcome

the obstruction and dilatation begins, the heart action becomes feeble and intermitting. The patient is subject to attacks of vertigo and syncope; there are great muscular prostration, marked pallor of the face, with marked anginal symptoms after or during excitement. In such cases sudden death may occur by a complete arrest of the ventricular systole.

Mitral stenosis admits of but slight compensation. If extensive, it is always a grave disease. The prognosis must be determined in every case by the severity of the thoracic symptoms. When during active exercise the lungs become congested and œdematous, with pulmonary hemorrhages, extreme dyspnœa, and cyanosis, the prognosis is especially unfavorable. Statistics furnished by Bellevue Hospital show that sudden death occurs as often in mitral stenosis as in aortic insufficiency.

Congenital mitral stenosis rarely causes death, and is attended by few cardiac symptoms except the murmur which characterizes it. The later in life the stenosis is developed the more unfavorable the prognosis. Cerebral embolism is one of the dangers of this valvular lesion.

Mitral incompetency is more often compensated for than any other valvular lesion. The changes which lead to its development occur slowly, and their tendency is to remain stationary except in young children. Patients with moderate insufficiency suffer very little except during or after violent exercise, and were it not for the slight dizziness which often follows such exertions it would pass unnoticed. In all cases where the compensation is perfect it is unnecessary to tell the patient that he has heart disease, for in no other valvular lesion is he so likely to live to old age without suffering any inconvenience. When, however, the compensating hypertrophy of the right heart gives place to dilatation and fails to overcome the obstruction in the pulmonary circulation, dyspnœa, cyanosis, with disturbances of the systemic circulation and œdema of the extremities, mark the beginning of the end. No valvular lesion in children is so serious as that form of mitral insufficiency caused by extensive thickening and retraction of the segments of the mitral valve, so that they form a narrow band of hardened tissue around a large open mitral orifice. Freedom from pulmonary congestion or any marked disturbance in the hepatic and renal circulations after prolonged physical exertion or excessive fatigue, in one who has mitral insufficiency, may always be regarded as a favorable indication.

Treatment of Chronic Valvular Diseases.

Chronic valvular lesions have three periods of development: first, a period of perfect compensation; second, a period when the compensation begins to fail; third, a period of complete failure of compensation. During the first period no medical treatment is required. The individual must lead a quiet life, free from excitement and worry. His diet must be non-stimulating and restricted in quantity to the requirements of perfect nutrition. In eating and drinking he should obey the physiological laws of health. Alcohol and tobacco should be used sparingly, if at all. Physical exercise is essential, but it should never be excessive or prolonged to fatigue. There are no fixed rules as to bathing. Each individual should be governed by its effects. In most cases

sponging the surface of the body daily with cold or tepid water is of service, for it has a tendency to keep the skin active and stimulate the surface circulation. Sexual indulgence should never be excessive, and in some instances be entirely abstained from. The office of the medical adviser is to keep the patient from exertion, mental worry, and those things which disturb his nutrition. If there is a strong rheumatic or gouty tendency, the patient should take up his residence in a moderately warm, dry climate, one that is not liable to sudden changes of temperature. The first indication that a patient with chronic valvular disease is entering on the period of *commencing failure of compensation* requires the most careful consideration. Undoubtedly, the earliest and most positive sign of such failure is sudden attacks of dyspnœa after active physical exertion or on awakening from sleep at night. Now the patient should be made to understand his exact condition and the dangers which attend it. He should be told of the serious effects of sudden physical strain or emotional excitement in producing cardiac dilatation —that simple over-indulgence in eating or drinking or sexual excess may lead to serious cardiac insufficiency which will never be fully recovered from. His emotions, passions, and indulgences must be kept under perfect control. Carefully regulated physical exercise is important, but it should never be carried to fatigue, and should always be immediately followed by a period of perfect rest. All these indications can best be met in a quiet country life.

In addition to what has already been said about diet, it must be remembered that the stomach must have all the rest compatible with perfect nutrition. It is often difficult to combine both indications. Our aim must be to obtain the most perfect nutrition with the least tax on the digestive organs. Therefore, this class of patients should indulge sparingly in sugars, starchy vegetables, and animal fats. Their food should be largely nitrogenous and taken in small quantities at a time, so as not to embarrass the heart action. If relief from the dyspnœic attacks and the other signs of commencing heart insufficiency does not follow careful regulation of the diet and exercise and freedom from care and excitement, a mild mercurial course, combined with small doses of digitalis and some saline, should be resorted to, the patient remaining in bed during its administration. I have found 2 gr. hydrargyri cum creta, with ½ gr. of extract of digitalis and 20 gr. of Epsom salts, every four hours, the best combination for overcoming the commencing failure of compensation. The use of digitalis during this period requires the greatest care. Harm is often done by giving more than is required to regulate the heart action.

When in chronic valvular disease failure of compensation, indicated by dilatation of the heart cavities and feebleness of the heart walls, is established, the period for active interference is reached. There is no class of cases which require sounder judgment and more experience in their management than these. While each case must be studied by itself, there are certain general rules which may serve as guides in their management. The same general plan of treatment suitable before the failure of compensation should as far as possible be continued.

In aortic stenosis, as soon as failure in compensation is established, not only must absolute rest be enjoined, but the patient must assume as

much as possible a horizontal position. The rules in regard to restricted diet must be rigidly enforced and all active exercise prohibited. The surface of the body should be covered with flannel, and exposure to sudden changes of temperature avoided. Frequent massage will give relief to the sluggish cutaneous circulation.

The drug which most certainly increases the force of the cardiac systole and relieves the vertigo and tendency to syncope in this condition is strychnine, in doses of $\frac{1}{60}$ gr. administered before taking food in the morning and in the middle of the day. It will often stay the compensation for a long period. Under its influence the pulse loses its irregularity in force and rhythm and the signs of heart insufficiency disappear. The failing compensation of aortic incompetency is usually associated with very great cardiac enlargement and extensive arterial changes; consequently, for a long time there will be periods of temporary loss of compensation, which will be quickly re-established by regulation of diet, mercurial catharsis, and rest; but when the period of complete failure is reached the patient has entered upon the most serious condition of valvular disease which we have to combat, except tricuspid insufficiency. Rest in bed does not now give the same relief as in aortic stenosis; nevertheless, it is important to maintain it as far as possible, and all the other rules which have been given regarding diet, exercise, etc.; but strychnine will not so certainly or so safely increase the force of the cardiac systole as it does in stenosis. I have often observed that this class of patients are made worse by the use of strychnine, while digitalis not only increases the force of the cardiac systole, but slows the heart action and causes the pulse to become firmer, fuller, and more regular in rhythm. If the only object is to increase the power of the ventricular systole and increase the contraction of the peripheral vessels, the tincture is its most reliable form for administration. It may be given in doses ranging from 5 to 30 drops in the twenty-four hours, the amount in each case being determined by careful trial, the patient remaining in bed during the time. When it acts beneficially, the dyspnœa will be relieved in twenty-four hours and the urine increased in quantity. It may be given in large doses for a long period with benefit. I have now under observation a gentleman fifty-two years old, with cardiac incompetency and a very large heart, who has taken from 20 to 40 drops of the tincture of digitalis almost daily for ten years. If he omits it for two or three consecutive days, he suffers with palpitation and cardiac distress, which soon disappear when he returns to his digitalis. If in any case of aortic insufficiency a condition of high vascular tension is reached, nitro-glycerin will often give the most marked relief. One drop of the officinal 1 per cent. solution may be given every three or four hours.

The period of established failure of compensation in mitral disease is marked by pulmonary complications, by a rapid, feeble, irregular pulse, by scanty urine, constant dyspnœa, and dropsy. The evidences of anæmia become marked in dilatation of the right heart. An important factor in the failing compensation is greater or less obstruction to the venous circulation. Everything that taxes or interferes with respiration must now be carefully avoided, and the greatest care must be maintained in diet and exercise. All the predisposing causes of bron-

chitis and acute pulmonary affections must be guarded against. Straining at stool must be avoided and the use of alcohol and tobacco prohibited. Gentle daily evacuation from the bowels must be maintained. These patients should avoid prolonged use of the voice, especially in speaking and singing.

The most strikingly beneficial effect of digitalis in the treatment of valvular lesions is seen in cases of mitral disease. Our aim in the administration of digitalis in mitral disease is to slow the pulse, make it more regular and of higher tension. The power of digitalis to strengthen the contraction of the right ventricle when it has become dilated is even greater than its action upon a dilated left ventricle. By its action on the right heart it overcomes the obstruction in the pulmonary circulation, and thus tends to restore the failing compensation of the left heart. It often does this in a surprising manner, causing the most extreme dyspnœa to disappear by relieving the over-distended pulmonary vessels and for a time re-establishing complete compensation.

In the administration of digitalis in mitral disease we should strive to obtain the desired therapeutic effect with the smallest possible dose. Five or ten drops of the tincture twice a day are often amply sufficient to accomplish all that is desired.

When it is important to produce a rapid and powerful effect in cases where there is considerable dropsy, marked embarrassment of the respiration, cyanosis, scanty urine, a small irregular pulse, and a tumultuous action of the heart, much larger doses may be given. Twenty drops of the tincture or half an ounce of the infusion every two hours for twenty-four or forty-eight hours may be required to accomplish the desired result. Whenever in mitral incompetency complete asystolism is present and suppression of the urine is threatened, digitalis should be given freely, whether the other indications for its use are present or not. In some mitral stenoses, however, even when the pulmonary symptoms are urgent and the cyanosis is extreme, digitalis should be used with great care, and its administration should be preceded by the free application of wet cups to the chest and leeches over the liver.

Whenever large doses of digitalis are being administered in any valvular disease it is important that the patient should remain in bed and the bowels be kept freely open with mercurial cathartics. Sometimes, when large doses of digitalis are prescribed in mitral disease for a considerable time, the toxic effect of the drug is produced. The pulse becomes irregular and small, there may be two beats of the heart to one of the pulse, the urine will be reduced in quantity, and there may be nausea and vomiting. The urine is the best guide as to the continuance of its administration: as long as it is increasing in quantity or freely secreted there is little danger of its toxic effect. If toxic symptoms occur, there is no danger attending it, for they soon disappear after the withdrawal of the drug.

In some cases of mitral disease, where the administration of digitalis is followed by beneficial results, it does not regulate the pulse.

In tricuspid incompetency secondary to mitral disease, where there are cyanosis and symptoms which indicate engorgement of the right heart, digitalis should not be administered until the venous engorgement

is relieved by venesection, by the application of leeches, or by free purgation, for digitalis and general stimulation are dangerous under such circumstances, and may destroy a life that might have been saved. There is nothing more harmful to patients with commencing failure of compensation in tricuspid insufficiency than the free use of digitalis.

There are certain pronounced symptoms of chronic valvular diseases which demand special consideration:

(1) *Dyspnœa.*—This is perhaps one of the most distressing symptoms of established failure of compensation. In those cases in which digitalis fails to re-establish the compensation and the attacks of dyspnœa are frequent and extreme or continuous, morphine administered hypodermically is invaluable and should be given without hesitation. It usually gives immediate relief; the amount to be administered in any case can be determined only by trial.

To obtain relief in advanced cases large doses may be necessary. In cases with extensive arterial changes or where there is high tension pulse a 1 per cent. solution of nitro-glycerin may give relief when administered in increasing doses in connection with digitalis. If the dyspnœa is increased by the presence of fluid in the thoracic or abdominal cavity, it should be promptly removed by aspiration. In all cases rest in a bed should be provided, which will support the arms and head and at the same time give rest to the feet and legs. Nitrite of amyl seldom gives even temporary relief to this class of patients.

(2) *Dropsy.*—Œdema of the feet is usually the first indication of commencing failure in valvular diseases. At the onset it usually rapidly disappears by the free administration of digitalis and rest in bed. If this fails, calomel and squill, combined with digitalis in the form of the Fothergill pill, composed of calomel, squills, and digitalis, each 1 gr., every four hours, will cause it to disappear promptly.

Hydragogue cathartics are of doubtful efficacy in the treatment of these cases, and the so-called diuretic plan of treatment has not been at all satisfactory in my hands. It is never advisable to attempt to remove the dropsy by hot air baths or pilocarpine.

In the advanced stage, when the patient's condition is rendered distressing by general anasarca, scarification of the skin will allow the serum to drain out and give the greatest relief. If it is done antiseptically after thoroughly cleansing the parts, it will not be followed by any unpleasant results. After the scarification the parts should be covered with carbolized cotton. The position of the patient with extensive œdema of the surface should be changed often, so that the dependent parts may not become greatly distended.

HYPERTROPHY AND DILATATION OF THE HEART.

By WARREN COLEMAN, M. D.

CARDIAC HYPERTROPHY and cardiac dilatation coexist in the same heart in the great majority of cases, the latter being the final effect of the causes which have induced the hypertrophy. As in other muscles of the body, there is a limit to development in the heart, after which degeneration ensues. The cause of this degeneration lies in the interference with the nutrition of the heart wall, either because the increase in the bulk of the cardiac muscle is so great as to produce not only a relative but an actual diminution in the quantity of blood supplied to the heart, or because atheromatous changes in the coronary arteries diminish their calibre and thus affect nutrition. Attention was directed to a similar failure of muscular power in the arms of file-cutters at Sheffield by Allbutt, and is known to occur also in the arms of blacksmiths, even though the muscles appear firm and well nourished. Thus we see that continued excessive use of muscles produces first hypertrophy with increased capacity for work, to be followed later by degeneration and failure of power.

It is generally conceded that hypertrophy may occur without dilatation, though even in these cases the hypertrophy is preceded by a dilatation which passes away. The demand for extra work on the part of the heart means increased intra-ventricular pressure, and this induces temporary dilatation. On the other hand, dilatation (not the transient form just mentioned) without a preceding hypertrophy is rare, but occurs at times when the heart is called upon to perform an undue amount of work, as in soldiers on a forced march, in inexperienced mountain-climbing, or when sudden exertion is made after one of the acute infectious diseases and the degenerated myocardium gives way, and under these circumstances may be a cause of sudden death.

HYPERTROPHY.

DEFINITION.—Cardiac hypertrophy is an increase in the thickness of the walls of the heart with or without alteration in the capacity of its cavities. It may be limited to a single chamber, to one side, or the whole heart may be enlarged. Hypertrophy of the left ventricle is by far the most common; next in order of frequency is hypertrophy of the left auricle; then the right ventricle; and finally, the right auricle.

Cardiac hypertrophy occurs in two forms—simple hypertrophy and hypertrophy with dilatation or eccentric hypertrophy. Fagge defines

simple hypertrophy as "an increase in the amount of the heart's muscle, its cavities remaining unaltered in capacity." Similarly, hypertrophy with dilatation may be defined as an increase in the amount of the heart's muscle accompanied by increase in the capacity of its cavities. A third form of hypertrophy was formerly described—concentric hypertrophy—a condition characterized by increased thickness of the wall of a cavity of the heart (usually the left ventricle), with diminution in its capacity. Most observers are agreed, however, at the present time that such an appearance is artificial. It is probably produced by the stopping of the heart in systole, and is met with constantly after the administration of strychnine in large doses shortly before death.

ETIOLOGY.—Cardiac hypertrophy results in all cases from an increase in the amount of work performed by the heart, whatsoever may be the cause of this increase, and provided the demand for it be long continued.

For sake of convenience of description we may divide the causes of cardiac hypertrophy into (1) causes in the bloodvessels, both systemic and pulmonary; (2) causes in the heart; (3) causes in the nervous system; and (4) toxic causes, whether they modify cardiac innervation or act directly upon the heart muscle.

(1) *Causes in the Bloodvessels.*—When for any reason there is interference with the flow of blood through the small arteries, the blood pressure rises and the cardiac contractions are increased in force to overcome the obstacle thus interposed. This interference may be due to inelasticity and loss of contractile power in the arteries, to narrowing of their calibre from changes in their walls, or to actual obliteration of definite capillary areas, thus bringing about an increase in the peripheral resistance. When these changes occur in the systemic circulation the left ventricle becomes hypertrophied; when in the lesser or pulmonary, the right ventricle.

The most common form of cardiac hypertrophy is that met with in old age, and which affects both ventricles. Bizot called attention to this form in 1837, and stated that it occurs uniformly in all individuals. Charcot recognized the condition, but said it occurs only in certain subjects, and regarded it as pathological. Balfour, who has written on the subject more recently, agrees with Bizot in that it occurs in all cases with advancing age. He further states that this very fact oftentimes renders the heart better able to perform its work at seventy years than at an earlier period. The causes of this senile hypertrophy are supposed to lie in the loss of elasticity and rigidity of the arterial walls and in the obliteration of large numbers of capillaries throughout the body. In opposition to the theory that obliteration of capillary areas will give rise to cardiac hypertrophy it is objected that such hypertrophy does not occur after amputation at the hip-joint. But under these circumstances the circulation readjusts itself to the new conditions, provided the arteries be normal.

General arterial sclerosis, whether inherited or acquired, affords another example of cardiac hypertrophy resulting from increased peripheral resistance and heightened blood pressure. In a healthy state of the arteries the amount of work which the heart has to do is regulated to some extent by the depressor nerve. Impulses passing up this nerve

from the heart to the vaso-motor centre in the medulla cause dilatation of the arterioles and lowering of blood pressure. But in arterial sclerosis the walls of the small arteries are stiff and rigid, and do not respond to the vaso-motor impulses which govern the amount of blood in any organ at a given time. The arteries are unable to dilate when the vascular system becomes over full; the blood pressure rises, and the heart must put forth greater energy to overcome the resistance of the more tightly closed aortic cusps. Further than this, the calibre of the small arteries is actually encroached upon by thickening of the tunica interna, so that the capacity of the arteries as a whole is diminished.

Such a condition of the arterial system is associated chiefly with the fibroid or gouty diathesis and with the chronic interstitial form of nephritis. The space at our disposal will not permit a discussion of the etiological relations existing between arterial sclerosis and disease of the kidney. Sufficient to say that chronic interstitial nephritis is accompanied in almost all cases by changes in the vascular system which lead to the development of cardiac hypertrophy. It is claimed that another form of nephritis may cause cardiac hypertrophy, the acute or subacute form which complicates or follows scarlet fever. In these instances the hypertrophy cannot be explained by general vascular changes, at least not those of a sclerotic nature. In an analysis of 360 autopsies made at the Johns Hopkins Hospital, W. T. Howard, Jr., found 62 cases of hypertrophy associated with arterial sclerosis. Of these, 52 showed general hypertrophy and dilatation, and 10 hypertrophy of the left ventricle alone. The greater proportion of cases were between thirty and sixty years of age. Two of the conclusions drawn from this analysis are—that arterial sclerosis is by far the most common cause of hypertrophy of the left ventricle,[1] and that it is the most common of all causes of hypertrophy in subjects more than thirty years of age, except such causes as are in the heart itself. The influence of kidney disease is well shown in the fact that of these 62 cases, 38 presented evidences of chronic diffuse nephritis.

Extensive atheromatous changes in the larger arteries also lead to cardiac hypertrophy. Normally, the intermittent outflow of blood from the heart is converted into a continuous flow in the capillaries by the over-distention of the arterial system, producing constant pressure from behind. When from atheromatous changes the arterial walls become rigid and inelastic the tendency is for the flow in the capillaries to become intermittent, though this never actually occurs. However, since the *vis a tergo* is diminished, blood accumulates in the venous system and offers an increased resistance to the arterial flow. As a further element in the production of this increased resistance it must be remembered that extensive atheroma in the large arteries is usually accompanied by similar changes in the arterioles. According to Axel and Stokes, aneurysm of the aortic arch produces cardiac hypertrophy only when associated with atheromatous changes in the arterial walls. Since, however, loss of elasticity in the aorta is not an infrequent cause, at least predisposing, of aneurysm of the aorta, the two conditions will often be found associated.

[1] It should be added, however, that the hypertrophy resulting from old age is not taken into consideration.

A condition of the circulatory system called *hydræmic plethora* has been described by Bamberger and by other observers as a cause of cardiac hypertrophy. It is met with most frequently in men who work around breweries and who consume large quantities of beer a day. The amount of liquid taken into the body is so much greater than is required for its uses, and is taken so constantly, that the kidneys are called upon to excrete greatly increased quantities of water. Bamberger states that in response to this demand the kidneys become hypertrophied. But sooner or later they are taxed beyond their power, more liquid is ingested than can be eliminated, the bloodvessels become filled beyond their normal capacity, and the blood pressure rises. As we have seen above, a continuously increased blood pressure throws more work upon the heart and necessitates its hypertrophy. In these cases, however, the alcohol contained in the beer and the amount and character of the work which many of these men have to perform must not be left out of consideration as etiological factors.

Congenital hypoplasia of the arterial system, such as occurs in chlorosis, is another cause in the bloodvessels of cardiac hypertrophy, in that it offers an increased resistance for the heart to work against. Congenital narrowing of the first portion of the aorta or compression of the aorta by mediastinal and other tumors—*e. g.* large abdominal tumors—may be mentioned as rare causes in the systemic vessels of hypertrophy of the left ventricle.

The principal causes in the lungs of hypertrophy of the right ventricle are emphysema and pulmonary fibrosis, whether of tubercular or other origin, in which conditions large areas of the pulmonary capillaries become obliterated. An exudation into the pleural cavity may act in a similar manner by compressing the capillaries. Atheromatous changes in the pulmonary artery or compression of the artery by mediastinal tumors and aortic aneurysms also cause hypertrophy of the right ventricle.

The hypertrophy of the heart which occurs during pregnancy is physiological in nature and disappears when lactation ceases, unless pregnancies follow each other in rapid succession, when the heart may become permanently enlarged. The increase in weight is about one fifth of the normal heart weight, and the enlargement is confined to the left ventricle. The etiological factors which induce this hypertrophy are probably increased frequency and increased force of the contractions incident to the demands of the pregnant state.

(2) *Causes in the Heart.*—Aortic stenosis and aortic regurgitation, either alone or conjointly, are the valvular lesions which produce the highest grade of cardiac hypertrophy. Under these circumstances the left ventricle only is affected, until, from changes in the mitral valve of an interstitial or atheromatous nature, or from weakening and stretching of the auriculo-ventricular ring, incompetency of this valve occurs and blood is forced back into the auricle. In either stenosis or regurgitation at the aortic orifice the left ventricle has to perform an abnormal amount of work. In the former case the blood is forced through the obstructed orifice with difficulty; in the latter the ventricle has to expel an abnormal quantity of blood.

Mitral regurgitation of organic nature is followed in all cases by

hypertrophy of the left ventricle, the cause being the increased amount of blood thrown into the ventricle at each auricular systole. This is the most common cause in the heart of cardiac hypertrophy.

Stenosis at the mitral orifice and mitral regurgitation cause hypertrophy of the left auricle. This hypertrophy is always accompanied by dilatation.

The most frequent causes in the heart of hypertrophy of the right ventricle are mitral regurgitation and mitral stenosis. These lesions act by raising the blood pressure in the pulmonary vessels. Among the rarer causes may be mentioned pulmonary stenosis and regurgitation.

General cardiac hypertrophy, or enlargement of all the chambers of the heart, is usually the result of incompetency at more than one valvular orifice. There may have been, however, only one primary lesion —as, for example, at the mitral valve—yet after the hypertrophy has reached a certain grade the tricuspid valve becomes incompetent from weakening and stretching of the auriculo-ventricular ring, so that the flaps of the valve cannot be approximated during the ventricular systole.

As has been stated in the section on Adherent Pericardium (p. 368), cardiac hypertrophy is a frequent sequel of pericarditis with adhesions, especially when the adhesions are both internal and external to the sac.

Active physical exertion continued over a long period of time is a recognized cause of cardiac hypertrophy. It is met with frequently in athletes, soldiers, laborers, and others whose vocations or avocations necessitate prolonged physical exercise. These cases have been called primary or idiopathic hypertrophy. In many of them the abuse of alcohol undoubtedly enters as an etiological factor, but in others—*e. g.* athletes and peasants—the only history that can be elicited is that of long continued and excessive use of the muscles. Stress has been laid also upon the previous nutritive condition of the patients, it being claimed that hypertrophy is met with much more frequently among the lowest classes, those who perform the heaviest labor and who subsist largely upon carbohydrate foods. While admitting the influence of bodily nutrition upon the heart, this is thought to be a more important factor in the production of dilatation than of hypertrophy.

For the reason of the hypertrophy which follows prolonged physical exertion we must turn to the physiology of the circulation. It is a well-known fact that exercise increases the amount of work which the heart has to perform. The explanation commonly offered is that the muscles, as the other organs of the body, demand more blood when in a state of functional activity than when quiescent, and that the heart action is increased to supply the additional quantity. Just what means of communication exists between the muscles and the heart is an open question. It has been suggested that the carbon dioxide liberated at each contraction of the muscles accumulates in the blood and acts reflexly upon the heart through the respiratory centre in the medulla. But, as Forster explains, if such were true, the blood in the aorta should contain more carbon dioxide than normal, which is not the case unless the exercise be carried to a dangerous limit. Muscular metabolism is a complex process, and not as yet thoroughly understood. In the absence of any demonstrated nerve connection between the muscles and

the heart physiologists assume that a substance or substances are generated during muscular contraction which, having entered the circulation, act in some manner, either directly or reflexly, upon the heart, increasing its activity. However this may be, the cardiac contractions are increased both in frequency and force, and, despite the dilatation of the arterioles in the muscle-areas, the blood pressure rises. If, then, as in the case of athletes and in the case of day laborers whose work requires constant muscular effort, such a condition of the circulation is kept up for several or many years without sufficiently long periods of rest, cardiac hypertrophy must result as certainly as when the demand for extra work is at one of the valvular orifices, or when the increased heart action is the effect of disturbed innervation.

In the case of violent and sudden straining the blood pressure rises still higher, because of the interference with the entrance of blood into the vessels contained within the thoracic cavity. The aspiration normally effected by inspiration is suspended; consequently, blood accumulates rapidly in the veins. And when the strain is over the right heart has to unload the venous system of its excess of blood.

Cardiac hypertrophy from over-exertion occurs in animals as well as in the human subject. The heart of the greyhound Master McGrath (Houghton, cited by Osler) weighed three times the normal proportion of that organ to the body weight.

DaCosta has described cardiac hypertrophy from over-exertion in its early stage as "irritable heart."

Finally, hypertrophy of the heart may be caused by acute dilatation of a ventricle during prolonged exertion or sudden straining. Unless death follows in these cases the balance of the circulation is ultimately restored by the development of a compensating hypertrophy.

It has been claimed that fibrotic changes in the myocardium (chronic myocarditis or cardiac fibrosis) may be the direct cause of cardiac hypertrophy—that the destruction of muscle fibres by the encroaching connective tissue necessarily leads to hypertrophy in other regions of the heart's wall. While this supposition must be admitted as possible, it is contended that such hypertrophy can rarely, if ever, be detected, and that actual enlargement never occurs from this cause alone. Cardiac fibrosis is the result of degenerative changes in the coronary arteries. The coronary arteries are not often affected with atheromatous changes independently of similar changes in other vessels of the body, and in the majority of cases follow them. This being true, the hypertrophy and the fibrosis appear to be the outcome of the same cause—in the one case the direct, in the other the indirect.

(3) *Causes in the Nervous System.*—Long continued disturbance of cardiac innervation, with accelerated and more forcible heart action, results in hypertrophy of the heart. Such hypertrophy is met with in patients who have been for a long time the subjects of cardiac palpitation, in those who have been addicted to excessive venery, and in cases of exophthalmic goitre. It is an interesting fact that the hypertrophy resulting from exophthalmic goitre disappears when the disease has been relieved.

(4) *Toxic Causes.*—These causes act chiefly by modifying cardiac innervation in such a way as to increase the force and frequency of the

contractions. The principal among them are alcohol, coffee, tea, and tobacco. Hearts hypertrophied from the abuse of alcohol, aside from an associated fibroid diathesis, are met with most frequently in men who are employed in breweries and who consume large quantities of beer. But the hydræmic plethora induced by the large amounts of liquid ingested and the influence of the amount and character of the work which they have to perform must not be overlooked as factors in its production. Symptoms of this form of hypertrophy—or, more accurately, symptoms of failing compensation—manifest themselves in these men toward the middle period of life.

Sex has no influence in the development of cardiac hypertrophy, except in so far as men or women are more subject to the respective causes. Cardiac hypertrophy is rare in childhood except as the result of valvular lesions or pericarditis with adhesions.

PATHOLOGICAL ANATOMY.—The size of the heart depends upon the age, sex, and development of the individual, other things being equal, and before forming an opinion as to whether a heart is hypertrophied these matters must be taken into consideration. Virchow's comparison of the size of the normal heart to the size of the fist holds good only in a general way, and relates to the fist of the subject not of the pathologist. Moderate grades of hypertrophy cannot be determined in this manner.

An hypertrophied heart is an enlarged heart, and the degree of enlargement necessarily corresponds to the grade of hypertrophy. In many cases, however, the size of the organ depends quite as much upon the amount of secondary dilatation.

Since, as we have seen, the heart becomes hypertrophied with increasing age, enlargement under these circumstances must be regarded as one of the normal physiological processes of old age. Hence only such hypertrophies as may be considered pathological will be described.

The weight of the heart is the only reliable evidence available in many cases of an increase in its size. This is especially true of the milder grades of hypertrophy. Measurements of the heart's walls are often inaccurate and lead to erroneous conclusions, for it must be remembered that many of the cases which come to the autopsy table have died of acute infectious processes attended by more or less degeneration and softening of the myocardium. Often in these cases after the heart has been opened the walls seem to become thinned under the most delicate handling. When the heart has stopped in systole the wall of the left ventricle particularly may appear enormously thickened, yet after a time, and especially after placing the heart in water, it returns to normal thickness. Further than this, when marked dilatation accompanies the hypertrophy the measurements of the walls may be nearly normal, and yet the weight of the heart be considerably increased. In well marked cases of hypertrophy, however, there will be little difficulty in determining its presence.

For the sake of completeness the measurements of the normal heart are given below. The following table has been arranged by Peacock:[1]

[1] *Reynolds' Syst. Med.*, vol. iv.

	Males.			Females.		
Thickness.	Lines.	m.m.	Inches.	Lines.	m.m.	Inches.
Walls of the right ventricle, base . .	1.85	4.16	.164	1.85	4.16	.164
Walls of the right ventricle, midpoint .	1.98	4.35	.176	2.	4.5	.177
Walls of the right ventricle, apex . .	1.42	3.19	.125	1.3	2.92	.118
Walls of the left ventricle, base . . .	5.15	11.58	.425	4.9	11.02	.432
Walls of the left ventricle, midpoint .	6.	13.15	.532	5.6	12.6	.497
Walls of the left ventricle, apex . . .	2.4	5.4	.214	2.5	5.62	.222
Septum between ventricles	5.73	12.89	.51	4.7	10.57	.421

It is necessary to let the heart become thoroughly relaxed before taking any measurements of its walls. As an additional word of caution, it may be added, do not include the base of a papillary muscle in the thickness of the ventricle. Except in those cases where the heart has stopped in systole any decided excess over the above measurements may be taken as evidence of hypertrophy.

The weight of the heart varies within normal limits. The average weight for the average male adult is from 9 to 11 ounces: for the average female, from 8 to 10 ounces. Any marked increase over these weights can result only from hypertrophy or neoplasms of the heart wall. It is perhaps needless to add that before weighing the heart all its cavities should be opened and any clots or uncoagulated blood removed. Hearts weighing from 15 to 20 ounces are not infrequently found, but greater weights than these are rarely met with independently of valvular lesions or chronic interstitial nephritis. The highest grades of cardiac hypertrophy occur with lesions of the aortic valves or with combined aortic and mitral valvular disease. Indeed, under these circumstances there seems to be scarcely any limit to the development of the heart. Such enormous weights as 64, 57, and 53 ounces have been recorded by Stokes, Alonzo Clark, and Beverly Robinson respectively.

Simple hypertrophy affects the left ventricle more frequently than any other chamber of the heart. The shape of the organ as a whole is but little altered except for a broadening of the apex. When the right ventricle is hypertrophied there is a marked increase in the transverse diameter of the heart. In uncomplicated mitral stenosis the discrepancy between the greatly enlarged right ventricle and the small left ventricle is very apparent.

In general cardiac hypertrophy all the diameters of the heart are increased—the organ loses its conical shape and becomes more or less ovoid. Hypertrophy of the papillary muscles always accompanies any decided increase in the thickness of the ventricular walls, though they may appear shortened from the lengthening of their transverse diameter. The chordæ tendineæ also become thickened and stiff. Hypertrophy of the auricles does not occur without a coexisting dilatation. In auricular hypertrophy the enlargement of the musculi pectinati is very marked at times.

The wall of an hypertrophied heart which has not undergone degenerative changes is of a deep red color, of tough consistency, and cuts with increased resistance. When degeneration is present the wall is pale.

New connective tissue developments not infrequently complicate cardiac hypertrophy. They occur most often and in greatest abundance in the interventricular septum, and appear as light streaks or patches upon a darker background. They are readily visible to the naked eye. In the majority of instances where any considerable amount of connective tissue increase is present the coronary arteries will be found to have undergone atheromatous changes. Hypertrophy of the heart associated with marked connective tissue increase has led to the term "false hypertrophy." As explained above, however, it seems advisable to regard the new connective tissue in the light of a complicating lesion.

It is believed that an actual hyperplasia of the cardiac muscle occurs in hypertrophy, and that the process is not simply an enlargement of pre-existing fibres.

Symptoms.—Hypertrophy of the heart, as of any other organ in the body, is a conservative process, and occurs in response to a demand for extra work. Its purpose may be stated to be the prevention of symptoms which otherwise would appear, or the relief of them after they have developed. This being true, cardiac hypertrophy presents no symptoms when the compensation is complete and when no disturbing factors interfere with the otherwise normal workings of the circulation. But, unfortunately, such factors make themselves manifest sooner or later. Changes begin in the myocardium, or the arteries, coronary, systemic, or pulmonary, become hard and inelastic because of the unaccustomed strain to which they have been subjected by the powerfully acting ventricles. The adjustment of the circulation to the conditions imposed is unstable and easily upset. Individuals who hitherto have hardly known that they possessed a heart now become aware of its existence from slight causes. There may be no symptoms whatever except on exertion or from emotional excitement. Shortness of breath comes on from ascending a flight of stairs or from attempting to take a little unusual exercise. Alcohol, coffee, or tobacco, even in what ordinarily would be considered moderation, causes unpleasant stimulation and a sense of fulness about the præcordium and in the vessels of the head and neck. There are frequent flushings of the face, flashes of light pass before the eyes, and noises are heard in the ears. The carotids may be seen and felt to throb when there is the least excitement. Often the eyes are bright and prominent, and the mucous membrane of the lips is redder than normal. Even a full meal may cause a disagreeable sense of oppresion in the epigastrium. But, as a rule, there is no cardiac pain. In certain cases the patients suffer from headache and vertigo; in others they become conscious of their heart action after going to bed. The pulse is full and strong and of high tension. Attacks of palpitation may, but they do not always, occur. A dry irritative cough may be the first symptom to lead the patient to consult his physician.

Any or all of the above symptoms may continue with the same or increasing severity until the myocardial changes become more marked and the symptoms and signs of commencing dilatation present themselves.

In this connection cerebral hemorrhages may be mentioned as one of the dangers of cardiac hypertrophy. When the arteries have become diseased and their resisting power diminished, any sudden increase of

force in the already powerful heart may cause rupture of an artery and extravasation of blood.

Physical Signs.—A physical exploration of the chest affords the surest indications of cardiac hypertrophy. Evidences of hypertrophy may be discovered in this manner long before any of the signs of failing compensation present themselves. When the physical signs are indicative of hypertrophy the diagnosis is positive, but they are often wanting even when the hypertrophy is advanced. They will vary with the extent of the hypertrophy, with the chamber or chambers affected, and with the condition and extent of lung substance intervening between the heart and chest wall.

Inspection.—Schrötter, following the lead of Skoda, contends that bulging of the præcordial region does not occur in hypertrophy except as the result of previous disease, such as pericarditis, which "softens the structures of the chest wall." This view has been opposed by Bamberger, Friedreich, Fagge, and others, and at the present time it is admitted by the majority of clinicians that extensive hypertrophy may produce bulging of the præcordium, especially in children and adolescents. Bulging of the intercostal spaces may be evident even though the chest wall does not arch forward.

The normal position of the apex beat is the fifth left interspace a little to the inner side of the nipple line. The apex beat in children is often in the fourth intercostal space, and in old people not infrequently in the sixth.

In general cardiac hypertrophy the apex beat is displaced downward into the sixth, seventh, or even eighth intercostal space, and carried to the left, sometimes as much as two inches beyond the nipple line. When the right ventricle alone or chiefly is enlarged, the displacement is to the left without much lowering of position, or there may be an epigastric impulse.

With extensive hypertrophy the impulse is visible over a greater area than normal, and is not so sharply defined against the chest wall. Moreover, the force of the impulse is seen to be increased, and a heaving of the chest wall is observed over several intercostal spaces.

Walshe states that a presystolic pulsation may occur in the region of the base of the heart when the auricles are much hypertrophied.

Violent throbbing of the carotids and vessels of the face may be noted in many cases of hypertrophy upon the least excitement.

Palpation.—By means of palpation we are able to detect the forcible character of the impulse often when it is not visible. The hand is distinctly raised by the impact of the heart against the chest wall, and hence the impulse is described as lifting or heaving .

Percussion of the heart is notably unsatisfactory in its results, yet it gives the surest indication of hypertrophy. There is an area of absolute dulness and an area of relative dulness.

The area of *absolute dulness* corresponds to that portion of the heart in actual contact with the anterior chest wall. It is roughly triangular in shape, bounded above by two diverging lines starting from the upper border of the fourth sterno-costal articulation on the left side and passing downward. The outermost line runs to the apex beat in the fifth left interspace, the other along the left border of the sternum to the

ensiform cartilage, the lower ends of these lines being connected by a third. The left side of this triangle is not often a straight line, because of the irregularity of the inner border of the left lung. And though a portion of the heart, uncovered by the lung, lies to the right of the left sternal border, still, on account of the intervening bone, it is difficult, if not impossible, to define its limits in that direction. The juxtaposition of the heart and the left lobe of the liver renders the cardiac continuous with the hepatic dulness, yet for practical purposes a line joining the upper end of the ensiform cartilage with the apex beat may be regarded as marking the lower border of the heart. A tympanitic element is often added to the percussion note when the left lobe of the liver is reached, because of the underlying and distended stomach, which still further obscures the lower limit of the heart.

The area of *relative dulness* lies above and to the left of the area of absolute dulness, and forms a border which shades off gradually into the normal pulmonary resonance. It corresponds to that portion of the heart which is covered by the anterior border of the left lung. The extent of this area in any given case usually varies with different clinicians, a personal element entering into its determination in that the transition from modified to true pulmonary resonance is indefinite.

Weil defines the area of relative dulness as follows: It begins above at the lower border of the third rib to the left of the sternum, and curves outward and downward, within the nipple line, to the apex.

Percussion of the area of relative dulness is chiefly of value in those conditions where the lung covers in the heart to a greater extent than normal—*e. g.* in emphysema.

Because of the relatively large size of the heart in children the areas of absolute and relative dulness are comparatively larger than in the adult. Dulness is almost constantly present to the right of the sternum.

In cardiac hypertrophy the area of dulness is increased vertically and transversely. The triangle of absolute dulness covers a greater extent of surface and the direction of its sides is altered. Its left side, instead of running within the nipple line, may cross it and extend for a distance of two inches to the left. Its right side may cross the sternum and pass beyond it for an inch or more, while its base will be directed obliquely downward and to the left. In such extensive grades of hypertrophy, and when dilatation is a marked feature of the case, the area of dulness loses its triangular shape and becomes somewhat quadrilateral, the fourth line taking the place of the left lower angle. The necessity for it is created by the marked rounding of the apex of the heart.

Fagge offers the following rule as a practical method of estimating the size of the left ventricle particularly: First mark out with an aniline pencil the left side of the triangle by running a line from the upper border of the fourth left sterno-costal articulation to the outer limit of the apex beat, then erect a perpendicular to this line from the upper end of the ensiform cartilage. He states that with a normal heart the first line should not exceed 2½ inches in length, and the second 1½ inches, though in extensive hypertrophy the former may measure from 4 to 5 inches and the latter 2 or more.

Dulness to the right of the sternum is indicative of hypertrophy of the right ventricle.

Auscultation gives in many cases even less positive results than percussion. The characters of the heart sounds will be affected more by the condition of the myocardium than by the extent of the hypertrophy. If there are no degenerative changes in the heart muscle and the hypertrophy be advanced, the first sound will be dull, prolonged, and booming. If, on the other hand, the nutritive condition of the muscle is poor, the sound may be scarcely audible. Bouillard has ascribed a metallic character to the heart sounds in hypertrophy, the so-called *tintement métallique*, but this is not present constantly either in the same or different individuals.

The intensity of the second sound depends chiefly upon the degree of arterial tension. When the tension is high the sound will be sharp and ringing. This increased intensity will be more evident over the aortic or pulmonary valves according as the systemic or pulmonary arterial tension is altered. Sometimes there is reduplication of the second sound from absence of synchronism in closure of the valves.

Diagnosis.—When cardiac hypertrophy follows lesions of the valves of the heart the diagnosis may be made in the majority of cases with little difficulty. On the other hand, when it is dependent upon renal disease or upon arterial sclerosis the diagnosis must be reached in certain cases by the history, the general condition of the patient, and the pulse, rather than by the physical signs, and is only probable at best. The evidences to be gained by a physical exploration of the chest may lead to entirely erroneous results. The typical signs of the affection may be wanting, and yet the heart be considerably hypertrophied; and, on the contrary, some of the signs may be present and the heart be normal in size. When, however, the physical signs are indicative of cardiac hypertrophy the diagnosis is positive.

The diagnosis rests largely upon displacement of the apex beat to the left without lowering of position, or to the left and downward; a heaving, forcible impulse; and an increased area of præcordial dulness of more or less triangular shape.

The normal respiratory act has but little influence upon the size of the area of cardiac dulness, but forced inspiration or expiration respectively diminishes and increases the area.

In obese individuals with thick thoracic walls it is often impossible to determine the location of the apex beat by the most careful palpation, and that after the body has been bent well forward in order to bring the heart and chest wall into closer contact. In such persons percussion of the heart also will be difficult. In females an excessively large mammary gland may seriously interfere with an examination of the cardiac region.

In other cases the shape of the thorax, particularly the so-called "pigeon-breast" shape, and the deformities resulting from kyphosis or scoliosis produce a relative displacement of the apex beat and render the definition of the cardiac limits by percussion difficult if not impossible.

In addition to these, alteration in the position of the lung from adhesions and lesions of the lung substance may occasion even more serious interference with the results of a physical examination. Contraction of the lung and retraction of the chest wall after pleurisy, em-

pyema, and pulmonary fibrosis, due to tuberculosis or other causes, may give rise to displacement of the apex beat and expose a greater portion of the anterior surface of the heart, necessarily increasing the area of absolute dulness. Or the area of absolute dulness may be increased by circumscribed pleuritic effusions and by consolidation of the anterior border of the lower lobe of the left lung. Consolidated lung tissue between the heart and chest wall may also increase the force of the impulse and intensify the heart sounds.

As the result of emphysema the apex beat may be displaced toward the right or the increased volume of the lungs may shut in the heart, diminishing the force of the impulse and decreasing the area of absolute dulness, even though the heart be hypertrophied. And in those cases where the lung becomes adherent to the anterior surface of the pericardium a resonance will be obtained on percussion where there should be dulness. In these cases the determination of the area of relative dulness will assist materially in the diagnosis.

Hence we see that all the evidences obtainable by a physical examination must be taken into consideration, and reliance not placed upon any one to the exclusion of others.

Displacements of the apex beat other than to the left or to the left and downward should arouse suspicion that some other condition than hypertrophy is present—some contraction or other process in the lung or pressure upon the heart within the thorax or from below the diaphragm. But it must not be lost sight of that in these cases hypertrophy may be present as well.

It remains to differentiate cardiac hypertrophy from the conditions with which it is most likely to be confounded.

Hypertrophy of the heart may be mistaken for pericarditis in the stage of effusion because of the increased area of præcordial dulness and its triangular shape, yet a careful inquiry into the physical signs will serve to separate the two conditions. In hypertrophy the force of the impulse is increased, and if it is displaced it is carried to the left and downward. In pericardial effusion the force of the impulse is diminished, and it is displaced upward and to the left. In pericardial effusion the abnormal area of dulness extends to the left of the apex beat, often as much as two inches, which is never true in hypertrophy. Such extension of dulness beyond the apex beat may be considered diagnostic of pericardial effusion. In hypertrophy the heart sounds are intensified, while in pericardial effusion they are muffled and indistinct and may be inaudible at the apex.

Tumors of the mediastinum sometimes simulate cardiac hypertrophy. They may cause displacement of the apex beat, may increase the area of præcordial dulness, and may intensify the heart sounds, but the displacement and the dulness they produce are not uniform, and will vary with the situation of the tumor. Moreover, the area of dulness is rarely triangular, such as we find in hypertrophy.

Prognosis.—Cardiac hypertrophy is a conservative process, and, except in those cases where it results from disturbances of cardiac innervation, is developed for the prevention or relief of symptoms. In so far its presence must be regarded as favorable. Osler divides the course of cardiac hypertrophy into three periods: that in which the hyper-

trophy is developing, that during which full compensation is maintained, and that in which compensation is failing. The last mentioned period commences with the secondary dilatation which ensues in all cases unless the cause of the hypertrophy be removed or the patient dies from some intercurrent affection, and it is at this time that symptoms appear in the great majority of cases. Cardiac hypertrophy, then, is not in itself dangerous. The danger lies in the subsequent dilatation. The rapidity with which this dilatation comes on varies greatly in different cases. By proper treatment—chiefly by regulation of habits, diet, and exercise—it may be warded off for years. In the great majority of cases the dilatation is dependent upon changes of slow development in the coronary arteries which affect the nutrition of the heart wall. But myocardial changes may occur independently of atheroma of the coronary arteries. The heart suffers with the body generally in states of malnutrition. Hence the prognosis is in a measure influenced by the physical condition of the individual, and dilatation is less likely to supervene rapidly in the robust than in the debilitated. The intercurrence of acute infective processes also affects the prognosis, in that they all are accompanied by parenchymatous changes in the heart of greater or less extent.

Not in all cases of hypertrophy is the period of full compensation reached. When acute dilatation occurs in one of the ventricles of the heart from rupture of a valve or other cause, the patient may not die immediately, but may drag on a miserable existence for months, and die ultimately from the failure of a compensating hypertrophy to develop.

When the cause of cardiac hypertrophy can be removed the hypertrophy ceases to develop and may in certain cases disappear—*e. g.* in pregnancy and exophthalmic goitre. The younger the patient, the more likely will be the arrest of the hypertrophy.

The cases of hypertrophy in which the prognosis is most favorable, except where the cause is removable, are those in which the hypertrophy develops slowly as the result of gradual changes in the bloodvessels or in the valves of the heart.

For the prognosis in the different forms of valvular lesions the reader is referred to the article on Chronic Valvular Diseases (page 399).

Treatment.—In every case of cardiac hypertrophy the cause should be sought for, and, if possible, removed. This relates more particularly, however, to the cases which are of nervous or toxic origin or which have resulted from over-exertion.

In any case better results are to be expected from hygienic than medicinal treatment. The tax upon the cardiac power must be reduced to a minimum. The bodily functions should be kept at the highest working level, and to this end the best possible state of nutrition must be maintained. The patient's diet must be regulated carefully both as to quantity and to quality of food. Over-eating is especially injurious. It is better for the patient to take small quantities of easily digested food at more frequent intervals. As little liquid should be ingested as is consistent with the perfect performance of the absorptive and emunctory functions. Alcohol, coffee, tea, and tobacco should be used sparingly or not at all, and, if used, the quantity should be gauged by their

effects upon the heart. An hypertrophied heart is especially responsive to these stimuli.

Gentle daily exercise is to be advocated as the best means of keeping up the muscular tone and of controlling the metabolic processes, but patients should be careful to avoid fatigue. The amount and kind of exercise must be determined for each case separately. All sudden or prolonged exertion, such as lifting, straining, running, etc., should be positively prohibited. A cold sponge bath each morning, followed by friction with a coarse towel, will be found beneficial in cases where it can be borne. The cutaneous circulation will be stimulated thereby and a more equal circuit of the blood maintained.

Tendencies to constipation should be overcome, if possible, by dietary measures, though an occasional saline purge acts well.

The patients should lead lives as free from care, anxiety, and excitement as possible.

In those cases of hypertrophy which result from general arterial sclerosis or chronic interstitial nephritis attention should be directed to maintaining the blood pressure at a lower level.

The treatment for this condition will be found under Arterial Sclerosis (page 547).

Medicinally, over-action of the heart is best controlled by aconite. DaCosta and Walshe both speak highly of this remedy. DaCosta advises the administration of 1 or 2 minims of Fleming's tincture or $\frac{1}{60}$ to $\frac{1}{30}$ gr. of aconitia three times a day; Walshe, $\frac{1}{8}$ gr. of the alcoholic extract, used similarly.

DILATATION.

CARDIAC DILATATION may be divided, from a pathological standpoint, into simple dilatation and dilatation with hypertrophy.

Simple dilatation is an increase in the capacity of a cavity or cavities of the heart without increase in the amount of the heart muscle, and is necessarily accompanied by thinning of the heart wall.

Dilatation with hypertrophy is an increase in the capacity of a cavity or cavities of the heart with increase in the amount of the heart muscle, and the heart wall may measure a normal or increased thickness. In the former case the increased bulk of the heart muscle is not apparent because of the dilatation.

Simple dilatation is always primary and acute; dilatation with hypertrophy is usually secondary and chronic, the exceptions being found in those cases of hypertrophy where the heart dilates acutely under sudden exertion because of degenerative changes in the myocardium—*e. g.* typhoid fever, diphtheria, or disease of the coronary arteries.

Moreover, these two forms of cardiac dilatation differ so markedly in their clinical manifestations that it seems advisable to consider them separately under the headings Acute Dilatation and Chronic Dilatation.

Chronic dilatation will be considered first, because of its relation to hypertrophy.

Chronic Dilatation.

Definition.—Chronic cardiac dilatation may be defined as a gradual increase in the capacity of a cavity or cavities of the heart, accompanied by hypertrophy of the heart wall.

Etiology.—The exciting cause in all cases of chronic cardiac dilatation is mechanical; that is, increased intra-cardiac pressure.

When the increase of intra-cardiac pressure is gradual, as in cases of obstructive and regurgitant lesions at the valvular orifices arising during acute or recurring endocarditis, the dilatation is slow, and in the majority of cases is compensated for by hypertrophy of the affected cavity. For years the hypertrophy overcomes the dilatation and increases a little in advance of it, unless the individual dies from some intercurrent disease, until an advanced grade of eccentric hypertrophy or hypertrophy with dilatation is reached. But a time will come when the hypertrophy no longer compensates, and when a further or secondary dilatation must take place. This time is measured in the majority of cases by the condition of the coronary arteries. Sooner or later they become diseased. Atheromatous changes begin in their walls and they dilate, or thickening of the tunica interna of the smaller branches occurs, diminishing their calibre. In either case the nutrition of the myocardium is impaired. Any violent exertion during this secondary dilatation may result in sudden death from over-distention and stoppage of the heart.

Pathological Anatomy.—The heart is enlarged, and, as a rule, more than one cavity is affected.

If the dilatation is marked, the increase in the size of the cavity or cavities is very apparent. Often the cavities contain a large amount of uncoagulated blood. If the heart has been acting feebly during the last few hours of life, the blood is likely to be coagulated, and the clots may be exsanguinated wholly or in part. The auricles may be distended with large clots, and clots often extend from the ventricles well into the aorta and pulmonary arteries.

In many cases the auriculo-ventricular ring of one or both sides is stretched, so that the tips of the valve flaps do not come into apposition, and often the difficulty of closing the orifice is still further increased by thickening of the free borders of the valves. In rare cases dilatation of the ring in the left heart is prevented by calcification at the bases of the mitral flaps. In a recent case such calcification had occurred without involving the body of the valves, and the ring remained practically of normal dimensions even though the left ventricle was markedly dilated and hypertrophied.

In speaking of the pathological anatomy of hypertrophy it was noted that the papillary muscles shared in the hypertrophy of the ventricular walls. This hypertrophy is compensatory, and prevents the powerfully acting ventricles from forcing the valve back into the auricle and causing regurgitation. When dilatation is commencing this hypertrophy of the papillary muscles plays a very important rôle in increasing the gravity of the condition. As long as hypertrophy alone is present, the length of the papillary muscles is such that after contracting they hold the valve flaps in a horizontal direction across the auriculo-ventricular

orifice. But when dilatation is established the papillary muscles and the thickened chordæ tendineæ are relatively shortened, so that when the systole has well begun the incompetency of the orifice is still further increased. This relative shortening and traction upon the valve flaps is very evident in hearts where marked dilatation has succeeded to marked hypertrophy. The more sudden the development of the dilatation, the more apparent will be the relative shortening of the muscles and chordæ tendineæ. It is met with in all cases of valvular lesions where toward the end of the scene comparatively rapid dilatation supervenes—where the muscle has met for years, perhaps, the demand for increased work, until from degenerative changes it is no longer able to respond.

The wall of a dilated heart, except in cases of acute dilatation, is usually pale and the coronary arteries in the majority of cases are diseased. The larger branches may be tortuous and dilated with evidences of atheromatous changes in their walls, or upon microscopic examination the smaller branches may be found partially occluded. Advanced cardiac dilatation is accompanied in all cases by passive congestions of almost every organ in the body. Since these conditions have been fully described under their appropriate headings, it will be necessary only to refer to them in this connection.

If the congestion of the lungs has been of sufficiently long duration, they will show the condition known as brown induration or cardiac pneumonia, which is, in substance, a fibrosis with extensive pigmentary deposits. They do not collapse entirely when the thoracic cavity is opened, and are tough and leathery in consistence. On section they are of a dark brownish or reddish color.

The liver is enlarged and presents the typical "nutmeg" appearance of passive congestion, the darker areas being the distended central veins with the adjacent portions of their contributing capillaries—the lighter, the peripheral liver cells with contained fat globules.

The kidneys are large and in many cases hard, so that the term "stony kidney" has been applied to them. Their color as a whole is darker than normal, and dark blood oozes from the cut surface. On close examination the congested glomeruli may be made out with the naked eye in many instances. Actual changes in the kidney substance are often present with the congestion. The spleen is enlarged and dark.

The vessels of the entire alimentary tract are distended with dark blood, and the stomach often presents the appearance of chronic gastritis.

Symptoms.—In considering the symptoms of chronic or secondary dilatation of the heart a distinction must be made at the outset between the early and the late stages of the affection. The history of almost every case of valvular disease is that of dilatation of the cavity behind the lesion with a compensating hypertrophy, which in most instances is complete for years. The dilatation is progressive in character, increasing slowly though steadily; yet the hypertrophy also increases, and with sufficient rapidity to overcome it. This compensation fails, however, in time, and symptoms present themselves.

It may be assumed that when compensation has failed the cavities of the heart are no longer able to empty themselves completely at the systole, and that a portion of their contents remains behind. Even

though the amount of blood retained in a cavity after each contraction be small, yet when the sum total of the contractions for twenty-four hours is taken into consideration it will be seen that interference with the circuit of blood must occur. And the greater the amount of blood remaining behind, the greater must be the circulatory disturbance. When failure of compensation first begins the symptoms will be mild, and will be present only when demands are made upon the heart for greater activity—*e. g.* during exertion. As the failure of compensation becomes more marked the symptoms increase in severity, though there are great individual differences as to the rapidity with which the condition advances. Patients may live for months in comparative comfort with such cardiac inadequacy that it seems the heart must fail to meet its demands. During the time when compensation is complete there are usually no symptoms, though the balance of the circulation is unstable. The symptoms of the early stage of secondary dilatation or commencing failure of compensation have been considered in connection with Chronic Endocarditis (pp. 388, 394), so that it remains to describe only the later manifestations or those which are chiefly the result of obstructed venous return, when from stretching of the auriculo-ventricular rings blood is dammed back from the ventricles upon the incoming stream. All the organs of the body suffer in consequence of the passive congestion thus induced. The blood is imperfectly aërated in the lungs, the brain receives a deficient amount of arterial blood, and the functions of the alimentary tract and abdominal viscera are perverted. Furthermore, the stagnation of the blood current permits the transudation of the liquid elements of the blood into the tissues and serous cavities of the body.

Usually the first symptom to attract the patient's attention is cardiac palpitation. It is accompanied in most instances by præcordial distress. The heart action becomes disturbed and the patient is painfully conscious of the disturbance. Moreover, it becomes disturbed from slight causes or without apparent cause. At these times the pulse is rapid, irregular, and, in the majority of cases, intermittent, and the patient's face is expressive of impending danger.

Dyspnœa is a prominent and distressing symptom. At first it comes on only after exertion, but later is present constantly, and the slightest movement increases the patient's discomfort.

The respiration is hurried and shallow, there is orthopnœa, and the speech is broken by pauses for breath. Cough and expectoration accompany the passive congestion of the lungs, and the sputum not infrequently contains blood.

The congestion of the stomach and intestines gives rise to dyspeptic symptoms, and in many cases to vomiting. The liver does not perform its function properly. The kidneys fail to excrete the normal amount of urine, and the urine itself is high colored, of high specific gravity, and often contains albumin, blood, and casts.

The œdema which accompanies cardiac dilatation usually begins in the feet and ankles, and may extend until the whole body reaches a condition of general anasarca. The serous cavities, the peritoneal, the pleural, and the pericardial frequently fill with the liquid elements of the blood, especially toward the close of life.

Physical Signs.—Since chronic dilatation is but a later stage of

hypertrophy, certain physical signs are common to both conditions. Important differences exist, however, which will be emphasized in the following account:

Inspection.—Dilatation of the heart is characterized by a feebleness of the apex beat which is in marked contrast to the forcible, heaving beat of hypertrophy. In those cases where general dilatation succeeds to general hypertrophy the displacement of the apex beat to the left and downward is not altered, though the impulse becomes visible over a greater area, extending sometimes as far as the axillary line. Moreover, the impulse is diffused, it being difficult to determine the actual point of impact of the heart against the chest wall. The impulse may possess a wavy character, which is probably caused by irregularity of contraction in the ventricular walls. When the right ventricle is much dilated it is displaced to the right of its normal position or actually into the epigastric region. Osler explains this phenomenon by supposing that the left ventricle is forced farther to the left under the border of the lung by the dilatation, and that the right ventricle only or chiefly strikes the chest wall.

Palpation.—The feebleness of the contractions becomes even more apparent when the hand is placed over the cardiac region. The undulatory character may be felt in many instances, though Walshe states that sometimes it may be seen when it cannot be felt.

Percussion.—The area of cardiac dulness is increased both laterally and vertically in general dilatation, and is more or less oval in outline. Dulness to the right of the sternum is constantly present when the right ventricle is much dilated. The direction of the increase and the limits of percussion dulness have been discussed under Hypertrophy (page 416).

Auscultation.—The first sound of the heart loses its muscular element and the valvular element becomes more pronounced. The dull booming quality disappears and the sound is short, sharp, and feeble, resembling the second sound. This condition is called *embryocardia*. The *gallop rhythm* of Osler also may occur. He compares the sounds of the heart to the "footfall of a horse at canter." As the dilatation progresses the sounds become fainter, and it may be difficult to distinguish the first from the second, or the second may be inaudible at the apex. It is stated that reduplication of the first sound occurs at times in advanced dilatation from asynchronism of contraction in the right and left ventricles. When murmurs have been present prior to the dilatation, auscultation often reveals nothing but a confused, more or less continuous, blowing sound, and under these circumstances it will be impossible to determine their rhythm. A systolic murmur or murmurs usually appear when the dilatation becomes marked from stretching of the auriculo-ventricular rings.

DIAGNOSIS.—When in a case of hypertrophy in which full compensation has been maintained signs of cardiac inadequacy, such as palpitation and vascular disturbances, manifest themselves, it marks the period of commencing dilatation, or at least of the myocardial changes preparatory to such dilatation; and when in these cases symptoms of venous obstruction are present in addition, the dilatation is exceeding the hypertrophy.

The diagnosis of cardiac dilatation rests mainly upon the following points: A diffused, feeble impulse; an increased area of cardiac dulness; and a short, sharp first sound, resembling the second sound in character. These signs, taken in connection with the signs of obstructed venous return, establish the diagnosis in the majority of cases.

The rules which have been given for the differential diagnosis of cardiac hypertrophy from pericardial effusion and intra-thoracic tumors will apply also in cardiac dilatation.

When a case is seen for the first time it will be necessary to determine whether the dilatation is in excess of the hypertrophy, in order to give a prognosis. In hypertrophy the heart strikes hard against the chest wall, giving the impulse a lifting, heaving character, while in dilatation the opposite condition obtains. The impulse is feeble and labored, and at times wavy or undulating, giving evidence of the difficulty with which the overtaxed ventricle contracts against the obstruction in front of it or expels the additional quantity of blood. The area of dulness is increased in both, but will be more quadrilateral in outline in dilatation. In hypertrophy the first sound is prolonged and booming, the second sharp and ringing; in dilatation both sounds are short and sharp, and it is sometimes difficult to distinguish the one from the other.

Prognosis.—The prognosis in chronic dilatation is always unfavorable. When the dilatation has once begun it can never be overcome. Its advance may be stayed for a time, but it is sure to become progressive sooner or later. So long as the action of a dilated heart is regular during ordinary exertion the prognosis is favorable as regards the duration of life. As a rule, it is not until its action becomes irregular and intermittent that marked circulatory disturbances occur.

Perhaps the most important factor which determines the rapidity with which dilatation progresses, when once it has begun, is the general state of nutrition. The lower the vitality, the less able will the heart be to perform its work; and since intercurrent or complicating diseases interfere with nutrition, they necessarily increase the gravity of the condition. The primal cause of the dilatation also influences the prognosis; thus the prognosis is better, as a rule, in cases which result from nephritis than in those which result from cardiac valvular disease.

When signs of obstructed venous return, such as constant dyspnœa, anasarca, alimentary and renal disturbances, become prominent, death will probably occur within a year.

Treatment.—The treatment of chronic dilatation of the heart is in the main that of cardiac valvular diseases, and will be outlined only in this connection. There are two chief indications to be met—to maintain the nutrition of the body at the highest level and to control the action of the heart.

The diet must be nutritious and concentrated, and food should be given in small quantities at frequent intervals. Iron should be given after meals. Moderate exercise should be taken daily, but fatigue must be avoided. All the functions of the body should be kept working normally. If the urine becomes diminished in amount, the kidneys should be stimulated by means of diuretics. The bowels should move each morning.

The drug which is most useful in controlling the action of the heart

is digitalis. Loomis stated that it may be given in full doses as long as the urinary secretion is increased in amount—that otherwise harmful results may follow. Caution must be exercised, moreover, in the administration of digitalis if there is reason to believe that the myocardium is fatty or fibroid. Strychnine may be given in conjunction with the digitalis. In many cases of secondary dilatation 5 to 10 minims of the tincture of strophanthus in 2 to 4 drachms of Basham's mixture will be found useful in the later stages of the affection.

In all cases of secondary dilatation special symptoms will arise from time to time and must receive appropriate treatment.

Acute Dilatation.

Definition.—Acute dilatation may be defined as a sudden over-distention of a cavity or cavities of the heart, accompanied by symptoms of serious cardiac embarrassment, and in certain cases quickly followed by death.

Etiology.—Acute dilatation results from increased intra-cardiac pressure.

The degree of dilatation in a given case depends upon the suddenness with which the intra-cardiac pressure is increased and the condition of the myocardium, other things being equal. It need scarcely be stated that a sudden and decided increase of pressure is more likely to prove serious in its effects than a gradual increase, or that a degenerated heart wall is less able to withstand an increase of intra-cardiac pressure than a normal one.

The blood pressure is maintained at a fairly constant average in health by means of the cardio-vascular nervous mechanism. As has been explained in the discussion of the causes of cardiac hypertrophy, a nervous communication exists between the heart and bloodvessels to the effect that when the heart action becomes labored the arterioles dilate and the peripheral resistance is diminished (see page 408). The pressure in front of the arterial valves of the heart remaining for the most part constant, the amount of energy which the heart has to expend to overcome this pressure is also fairly constant. And it follows that when the blood pressure either in the systemic or pulmonary vessels is raised the intra-cardiac pressure must be increased correspondingly in order to carry on the circulation.

A sudden increase of blood pressure is induced by severe muscular exertion, as in heavy lifting, mountain-climbing, difficult athletic feats requiring strength and endurance, and forced marching, and if such exertion is persisted in, after embarrassment of the heart has manifested itself, by one who is not in "training" or by a man in the middle period of life, serious dilatation of the heart is likely to follow. Under these circumstances the right ventricle is more often affected than the left.

Those forms of exertion which necessitate the excessive use of the muscles of the legs are followed most quickly by embarrassment of the heart. Lagrange explains this fact on the theory of an auto-intoxication with carbon dioxide. Since carbon dioxide is one of the products of muscular contraction, the greater the volume of muscles and

the greater the number of contractions, proportionately greater will be the amount of carbon dioxide produced. The muscles of the legs being large, the amount of carbon dioxide liberated during their contraction is also large. So that in addition to the increased work demanded of the heart, primarily on account of the muscular exertion as such, this additional quantity of carbon dioxide has to be eliminated through the lungs. Moreover, the inadequacy or suspension of breathing which attends such exertion interferes with the entrance of blood into the veins of the thorax, and the pressure in the veins without the thorax rises rapidly. The venous system becomes over full of blood highly charged with carbon dioxide on its way to the lungs to be aërated. And when the back pressure in the veins is relieved somewhat, the balance of the circulation is seriously disturbed, the right heart receives a larger quantity of blood than normal, the lungs become actively congested, and the heart has to put forth greater energy to overcome the obstruction thus offered. The action of the carbon dioxide on the vaso-motor centre in the medulla (when the exertion is carried to such extremes all of it is not eliminated in the lungs) still further adds to the embarrassment of the heart by causing contraction of the arterioles and increasing the peripheral resistance.

Such exertion is much more serious for a man in the middle period of life than for a boy or a younger man, chiefly for the reason of arterial changes. Even among boys and young men, whose vascular systems are responsive to stimuli from the vaso-motor centre or to reflex stimulation from the heart over the depressor nerve, athletic sports requiring excessive use of the muscles of the legs should be entered into with caution, and tests of endurance should not be made without thorough previous training. Cases of heart-strain arise in this manner, recovery from which may take months or years, and which in certain instances never occurs.

It is stated that during such severe exertion, when the dyspnœa becomes distressing and the embarrassment of the heart extreme, the auriculo-ventricular ring of the right heart dilates and the tricuspid valve fails to close the orifice, thus by a safety-valve action permitting regurgitation into the auricle and relieving the over-distended ventricle of part of its blood. It is by this means that actual dilatation is prevented when the exertion is carried to dangerous lengths, provided the individual does not stop sooner because of the dyspnœa. When actual dilatation of the ventricle has occurred, recovery can take place only by means of subsequent hypertrophy.

Sudden death may occur under these circumstances from acute dilatation of the right heart. The late Dr. Byron stated to me on one occasion that death from this cause was not infrequent during the war between Chili and Peru and Bolivia (1879–83) among young, undeveloped recruits.

Another instance of dilatation of a perfectly normal heart wall is seen after rupture of a cardiac valve from violent and prolonged straining. Fortunately, such accidents are rare, but they do occur. In these cases two streams of blood pour into the exposed cavity during diastole and cause an increase of intra-cardiac pressure at a time when the heart wall is least able to resist it. If the individual survives, the balance

of the circulation is ultimately restored by a compensating hypertrophy.

Any disease or condition which is associated with or causes degenerative or substitutive changes in the heart wall, such as the acute infectious diseases and syphilis or cardiac fibrosis, predisposes to dilatation. The sudden deaths which result in diphtheria, typhoid fever, scarlet fever, etc. from sitting up in bed undoubtedly are caused by over-distention and paralysis of one or both ventricles of the heart. A number of cases of sudden death from dilatation of the heart in pneumonia are to be found among my autopsy records at the Loomis Laboratory. The patients have dropped dead on the street or been found dead in cheap lodging-houses, having died during the night. Almost invariably the right heart has been found distinctly dilated and filled with dark, partially coagulated blood. These cases have come without exception from the lower classes, and evidences of the abuse of alcohol have been present in their appearance and in the findings at the autopsy. From the circumstances attending their death and from the typical lesion in the lung it may reasonably be assumed that the men had kept on their feet attempting to work or to walk (many of them having been tramps) until the right heart gave way. In these cases, however, the effects of alcohol upon the myocardium have to be borne in mind as well as the effects of the toxin of the disease.

Thrombosis of the larger divisions of the pulmonary artery may be mentioned as a rare cause of acute dilatation of the right ventricle.[1] Two such cases have been met with in the last few months at the Bellevue Morgue. One case was that of a man in whom large emboli had been dislodged from a cardiac thrombus, the other a woman who had given birth to a child at full term a month previously, and had become slightly septic. In both instances large divisions of the pulmonary artery in both lungs contained thrombi entirely occluding their lumina, and the right ventricles were found enormously dilated.

The sudden deaths which sometimes occur when persons plunge into cold water are explained by supposing that the contraction of the small vessels in the skin area raises the tension in the aorta to such an extent that the heart is unable to contract against it, and is paralyzed in the attempt.

Pathological Anatomy.—The heart is enlarged. As a rule, however, the enlargment is not uniform, one cavity or one side of the heart only being dilated. The affected cavity is not infrequently filled with dark, imperfectly coagulated blood, and its wall is thinned. The right heart dilates acutely more frequently than the left in previously healthy individuals, for the reasons that the obstruction to be overcome arises more frequently in the pulmonary circuit, and that the right heart is less able to meet demands for increased work because of the relative thinness of its wall.

If the obstruction has occurred in the systemic vessels, the left ventricle will be found dilated.

In none of the cases from which the facts here stated concerning acute dilatation have been drawn did dilatation of both ventricles occur.

[1] Reported by permission of Dr. W. Gilman Thompson.

The auricle of the corresponding side may be dilated as well as the ventricle.

Symptoms.—We have seen that acute dilatation of the heart is the result of sudden and violent or violent and prolonged muscular exertion; hence the onset of the symptoms is more or less rapid, varying, however, in suddenness according to individual peculiarities of constitution and "staying power" or respiratory training. An athlete, for instance, is capable of performing feats of strength and endurance in the attempt of which another man would fail utterly, and perhaps with serious heart-strain as the result. In the majority of cases the symptoms of acute dilatation are preceded by the phenomena which attend excessive physical exertion. These are at first quickened heart action and accelerated respiration, which give a healthy glow to the surface and produce a general sense of well-being. When the exertion is carried beyond these limits breathlessness comes on, and perhaps what is called a "stitch" in the side, usually the left. The breathlessness increases in proportion as the exertion is continued. The chest seems bound down as if by a constricting band. A sense of oppression is felt over the præcordial or epigastric region. The individual becomes dizzy and feels as if he would fall. Nausea may or may not be present. The heart action is rapid, the pulse is small and, it may be, irregular or intermittent. The skin, particularly of the face, assumes an ashy gray or leaden hue, while the lips are of a dark purple color. The vision becomes obscured and flashes of light pass before the eyes. The ideas become confused, the intellect clouded, and the man falls, perhaps unconscious.

All of the above symptoms may occur without actual dilatation of the heart, at least of a permanent nature, and after a few hours or a few days the individual returns to a normal condition. Even in these cases, however, cardiac palpitation and dyspnœa are likely to come on for some time afterward when exertion is made. On the other hand, dilatation may occur from which the individual recovers only after months or years, or in extreme cases death may result.

Allbutt's description[1] of his own experience while climbing mountains in Switzerland may be taken as an example of the milder grades of acute cardiac dilatation. After climbing all day he was seized with sudden dyspnœa and epigastric oppression. On placing his hand over his heart the impulse was found labored and diffused, and percussion gave evidence that the right ventricle extended beyond its normal limits. Rest in the recumbent position with the shoulders elevated caused the oppression and increased dulness to disappear. After a few minutes he was able to walk and even to run on level ground, but when he attempted to go higher up the mountain the symptoms returned. During the following night he was awakened by severe palpitation and dyspnœa, which were relieved by going to the window and taking a few deep inspirations.

The following case illustrates the principal symptoms where a more serious dilatation has occurred: The patient was a well-developed, muscular man. He had not been drinking or indulging in any excesses, and states that he had never felt better in his life. While ascending the

[1] *St. George's Hosp. Rep.*, cited by Fagge.

mound of the Belgian Lion (200 feet) he was seized with dyspnœa when about two-thirds of the way up, but continued the ascent, though more slowly. The breathlessness increased as he ascended; his feet became heavy and almost devoid of feeling. Præcordial oppression came on. When he reached the top of the mound every contraction of the heart was felt distinctly, and it seemed as if he had innumerable hearts all over his body—in his head, in his hands, in his legs—all beating synchronously. He suffered from vertigo and dizziness, and would have fallen but for the support of the railing. His respiratory difficulty became very great, and the præcordial oppression almost unbearable—a sensation as if some great weight were crushing the heart. Moreover, at each contraction the heart appeared to "roll over." The pulse was rapid, small, and irregular. The distress in the præcordium was somewhat relieved by bending toward the left side. There was no nausea nor did the patient lose consciousness. After resting a few moments the symptoms mitigated in severity, and he was able to descend the mound with caution. While in a coach on his way back to the hotel nausea appeared, and he became so faint that he had to lie down. The administration of stimulants restored him temporarily.

During that night the dizziness and nausea continued at intervals, but there was no vomiting. On the following morning the cardiac oppression was still present, and the dyspnœa became extreme upon the slightest exertion. The patient says he felt fully twenty years older than before the attack. The next day, while travelling in the cars, and without having made other exertion, the præcordial oppression became distressing and the dyspnœa and dizziness reappeared. The "rolling over" of the heart become painfully evident; the pulse became rapid, small, irregular, and almost imperceptible; the respiration sighing. The voices around him sounded distant; his pupils dilated and his extremities grew cold.

After a rest in bed of two days the dizziness and præcordial oppression disappeared. But for six weeks the patient could not walk rapidly without marked dyspnœa, and he ascended stairs with great difficulty. Even at the present time, one and a half years subsequently, he cannot undertake violent exertion without decided dyspnœa.

In the two cases above mentioned (page 429), where sudden death from acute dilatation of the right ventricle followed thrombosis of large branches of the pulmonary arteries, the patients did not live longer than five or ten minutes after the onset of the symptoms. The man was lying in bed, apparently comfortable; the orderly had just ministered to some want. A moment or two later he was gasping for breath, his pulse was imperceptible, and before the house physician could be summoned he was dead.

The woman was convalescing apparently. During three or four days immediately preceding her death she suffered from slight shortness of breath, and the latter part of the night before she died she was very restless. A little before six the following morning she sat up in bed to bathe her face, and a few minutes later, while taking a cup of coffee, fell back upon the pillow. The house physician was summoned, and he found the patient cyanotic and gasping for breath. Her pulse was

rapid, small, feeble, irregular, intermittent, and at last imperceptible. Stimulants were administered, but without avail, and she died a few minutes afterward. At the autopsy the results already detailed were found. (See page 429.)

The last two cases may be taken to represent the type of those where death succeeds immediately to acute dilatation either of the right or left ventricle. Such deaths sometimes occur from suddenly sitting up in bed during or after one of the acute infectious diseases, as typhoid fever and diphtheria, or when acute dilatation occurs in an hypertrophied and dilated heart during some unusual and violent effort.

Physical Signs.—The physical signs of acute dilatation of the heart are a feeble, fluttering, diffused impulse; an increase of cardiac dulness to the left if the left ventricle is dilated, to the right of the sternum if the right ventricle is affected; and weak, rapid, indistinct heart sounds.

Diagnosis.—The diagnosis of acute dilatation of the heart rarely presents much difficulty. These cases generally give a history of prolonged exertion or violent straining, with the sudden or gradual development of great circulatory disturbance. The most prominent symptoms are extreme dyspnœa; weak, irregular, intermittent heart action; and cerebral disturbances, with loss of consciousness in certain cases. Examination of the heart reveals a feeble and diffused impulse, an area of dulness to the right of the sternum (for, except in cases of previous cardiac disease, the right ventricle is usually affected), and feeble, indistinct heart sounds.

It is not always possible in these cases to learn at first whether serious dilatation has occurred or not, and we must wait until the effects of perfect rest can be observed before the diagnosis can be made. If actual dilatation has not occurred, the symptoms will pass away entirely, or will return in milder degree upon exertion for a limited time afterward. If actual dilatation has occurred, the symptoms will continue until death ensues or until such time as may be necessary for an hypertrophy sufficient to overcome the dilatation to develop. When acute dilatation is due to rupture of an aortic cusp, a diastolic basic murmur will appear in addition to the signs above mentioned.

Prognosis.—In considering the gravity of acute dilatation it must be borne in mind that sudden death sometimes occurs, and that even though death does not follow immediately in all cases, it may ensue from the failure of a compensating hypertrophy to develop.

A prognosis cannot be given in any case until the extent of the dilatation has been determined. This can be done best by observing the effects of complete rest in bed. If the heart regains its control over the circulation within a short period and cardiac symptoms do not return upon moderate exertion, a favorable prognosis may be rendered. If, on the contrary, the circulatory disturbances persist, the prognosis is unfavorable as regards the heart itself, though not necessarily unfavorable as regards the duration of life. Under appropriate treatment the heart may be able to perform its work until the balance of the circulation is restored by hypertrophy of its walls. But it is probable that the heart will always remain in a weakened condition in these cases, and be unable to meet excessive demands upon its power.

TREATMENT.—The most important indication in the treatment of acute dilatation is to diminish the work of the heart. This can be accomplished best through absolute rest. The patient should remain in bed, free from mental excitement, until the cardiac symptoms disappear. When the dilatation first occurs and the heart action is feeble and irregular, stimulants should be administered, and of these alcohol is probably the best. It should be given in doses sufficient to control the heart. If alcohol should fail in its action, amyl nitrite, nitroglycerin, sodium nitrite, or hypodermic injections of strychnine may be tried. An ice-bag or Leiter's coil applied to the præcordium will frequently afford the patient relief from the cardiac oppression and quiet the action of the heart. In certain cases, when the venous system is overloaded with blood which the right heart is unable to carry off, venesection is attended with favorable results.

After the acute symptoms have disappeared and the patient is allowed to get out of bed, the greatest caution should be exercised against over- or sudden exertion. He should be careful not to place himself in positions where such exertion may become necessary. He should not attempt to run or to ascend stairs quickly. Cardiac tonics and stimulants should be administered during this period with a view to assisting the action of the heart until the development of a compensating hypertrophy has been completed. The best remedial agent is digitalis—5 minims or more of the tincture three times a day, as the case may require: $\frac{1}{60}$ gr. of strychnine after meals will be found a valuable adjuvant. Quinine sulphate in tonic doses of 4 to 6 gr. a day will act both as a cardiac and general tonic.

CARDIAC ATROPHY; CARDIAC RUPTURE; ACUTE AND CHRONIC MYOCARDITIS; THE FAT HEART.

By ELBRIDGE G. CUTLER, M. D.

CARDIAC ATROPHY.

Definition.—Cardiac atrophy, atrophia cordis, is a condition in which we find a diminution or partial disappearance of the heart muscle.

Etiology.—Usually the affection includes the whole heart; less frequently only a part of it is involved. The latter is found in the left ventricle in stenosis of the left auriculo-ventricular orifice, probably because the left ventricle gradually accommodates itself to the small amount of blood which flows into it during diastole as the result of the narrowing.

Total atrophy of the heart is one of the changes of advanced age, for the heart, like most of the organs, undergoes in extreme old age senile atrophy. It is also found where the organism has suffered loss of some of its fluids and has got into a marasmic and cachectic condition —the cachectic cardiac atrophy so called. Carcinoma, tuberculosis, long continued suppuration, severe typhoid, dysentery, diabetes, losses of blood, stenosis of the œsophagus, and similar diseases lead to it.

Sometimes there is a sort of pressure atrophy of the heart. Such a condition is observed in mediastinal tumors, in long standing pericardial exudations, in indurated thickenings of the epicardium, and in excessive development of subepicardial fat.

Narrowing of the coronary arteries may result in atrophy of the heart muscle from deficient nutritive supply.

The congenitally small heart first described by Rokitansky as occurring in delicate women with amenorrhœa and accompanied by deficient development of the genital organs was called by Virchow hypoplasia of the heart, since it represented rather an arrest of development than an atrophy. Virchow also speaks of a hypoplasia of the heart as occurring in bleeders.

Pathological Anatomy.—An atrophied heart is characterized chiefly by its small volume, its thin walls, and light weight. According to Wunderlich, when the heart of an adult does not weigh two hundred grammes it may be considered atrophied. Engel found the heart in consumptives to be reduced in weight to one fourth of the normal, and that the diminution in weight affected the left ventricle chiefly.

The pericardium is frequently found filled with clear serum.

The heart appears folded and wrinkled on its surface, which is particularly well seen in places where the epicardium is thickened by white spots, and it then resembles a dried pear in appearance and shape. Usually the subpericardial fat has disappeared, and presents the appearance of a slimy or gelatinous mass. Much more rarely great increase of the subpericardial fat is found. The coronary arteries are often very tortuous.

The cardiac muscle is frequently pale, but hard and dense; its color resembles muscle which has been macerated a long time in water. In other cases it is colored rusty brown or dark ochre yellow, as is found in aged people, cancerous and phthisical patients. The reason of this color is found on microscopic examination to be due to an abundance of yellow and brown pigment granules in the muscle fibres, which sometimes are arranged in rows, and are especially collected at the poles of the muscle nuclei. They have been considered to be the remains of muscle coloring matter. These changes have been given the name of brown atrophy, pigment atrophy, or pigment degeneration of the cardiac muscle.

Under certain circumstances the microscopic examination shows other degenerative changes in the atrophic muscle fibres. Friedreich found in the atrophy following cancer or tuberculosis that the muscle fibres had lost their transverse striæ and become changed into homogeneous colorless cylinders, with partial loss of their nuclei. He designated these changes as sclerosing atrophy, because even microscopically the cardiac muscle was characterized by great density and smooth section, with a wax-like lustre and almost transparent grayish red appearance.

In other cases the muscular fibres of the atrophied heart are partly granular, partly fatty, and have undergone amyloid degeneration.

These secondary changes may perhaps indicate that the atrophy depends not only on a shrinking of the muscular fibres, but also on a partial disappearance of them through degenerative destruction.

The aortic valves not infrequently also exhibit atrophic changes, being thin and delicate, and perhaps even being perforated and penetrated. The free edge of the mitral and tricuspid curtains are often curled up.

The cardiac chambers usually appear smaller than normal, because they accommodate themselves to the ordinarily diminished quantity of blood. This has been called concentric cardiac atrophy. On the other hand, excentric cardiac atrophy has been spoken of when the cardiac lumen has been enlarged, while in simple atrophy the chambers are unchanged. Excentric cardiac atrophy is more rare, and is usually seen in aged people if there is no great diminution of the quantity of their blood.

Symptoms.—A series of symptoms have been given by means of which cardiac atrophy is said to be recognized. They are, however, partly theoretical, partly accidental, and are quite unreliable for diagnosis. Among the objective symptoms diminution of the cardiac dulness is to be expected, but is of course only available for diagnostic purposes when pulmonary emphysema can be excluded.

Diagnosis.—The diagnosis of cardiac atrophy can only be a probable one.

PROGNOSIS.—The prognosis is unfavorable, because serious disease lies at the bottom of the condition.

TREATMENT.—The treatment would consist in strengthening the heart and enjoining complete physical and mental quiet.

CARDIAC RUPTURE.

SYNONYMS.—Rupture of the heart; Spontaneous cardiac rupture; Cardiorrhexis spontanea.

DEFINITION.—Separation or laceration of the muscular fibres of the heart, usually accompanied by hemorrhage and followed by death.

ETIOLOGY.—Spontaneous rupture of the cardiac muscle only occurs when preceding changes have taken place in the muscular fibres which have diminished their firmness and resistance.

Rupture of the heart occurs most frequently in disease of the coronary arteries. Richard describes an aneurysm of the coronary artery which led to erosion of the muscle and ended in rupture. It is much more frequently due to occlusion of the coronary arteries by embolism or thrombosis, which produces abscesses or softening of the heart muscle.

Another cause of rupture is fatty heart.

Myocarditis may be the cause of the tear in the muscle. Abscesses of the heart and acute or chronic cardiac aneurysm resulting from inflammation of the fibres may be the occasion of the rupture not infrequently. In some cases a limited dry pericarditis causes circumscribed fatty degeneration of the cardiac muscle, followed by rupture. Such an instance has been cited by Buhl.

New growths and ecchinococci in the muscle have caused rupture of the heart.

Diseases of the valves, cardiac orifices, and great vessels which are accompanied by stenosis lead to rupture when a necrosis or fatty degeneration of the cardiac muscle has occurred. In this connection narrowing of the aorta at the point of insertion of the ductus Botalli is to be mentioned.

Rupture of the heart sometimes occurs unexpectedly during complete rest of body and mind or even during quiet sleep. At other times physical or mental excitement is the immediate precursor. It has been observed after the lifting of a heavy load, a fatiguing dance, a luxurious meal, during straining at stool, coitus, epileptic convulsions, and so forth. The rupture has occurred during the act of vomiting, during a paroxysm of coughing, in childbirth, and after delivery. The chill in intermittent fever or the taking of a cold bath may cause rupture of the heart when the cutaneous vessels contract, and thereby offer great resistance to the left ventricle. Rupture of the heart has been observed after relatively slight injuries, and also after great physical exertion.

The affection is more frequent in men than in women, and it is one of the diseases affecting the aged, being found usually after the sixtieth year.

Pathological Anatomy.—Anatomically, a complete and a partial cardiac rupture are recognized. Complete rupture consists of a solution of continuity in the whole thickness of the heart wall, while in partial rupture destruction of a few layers of muscle, trabeculæ, or papillary muscles only occurs.

Complete rupture most frequently affects the left ventricle, because this is the part which oftenest suffers the preceding changes in the heart muscle which produce it. And here, again, it is the anterior wall of the ventricle near the heart's apex where it is usually seen, and not rarely it encroaches on the lowest part of the wall of the septum. It is rare to find the rupture on the posterior wall of the ventricle. Next in frequency is rupture of the right ventricle, more rare is rupture of the ventricular septum, next comes rupture of the right auricle, and, rarest of all, is rupture of the left auricle.

Both the endocardial and epicardial openings of the tear are characterized by their irregular jagged course. Sometimes the former, sometimes the latter, is the greater. Often both openings are plugged by clots. The length of the rent seldom exceeds one centimetre, but there are records of its including the whole height of the ventricle. The rent follows the course of the muscular fibres and layers, so that it forms a tortuous, fistulous track, with the outer and inner openings not opposite each other. It is rare for the rupture to be across the fibres, such cases being specially dangerous. It may happen that the fibres in the auricles instead of being torn are separated.

Usually there is only a single rupture, but it sometimes happens that the original simple track divides peripherally and opens on the epicardium into several tears. Of course those cases must be excluded from this category in which the epicardium bridges over a rupture.

The muscular layers of the heart adjoining the rupture are undermined, shredded, and more or less infiltrated with blood. Hemorrhage into the pericardial sac almost always follows. Sometimes this is very great and rapid; sometimes it gradually leaks out. It may amount to several pounds, so that on removal of the sternum after death the pericardium is tense and greatly distended by blood. If, however, there have been pericardial adhesions, as occur in chronic cardiac aneurysm, the hemorrhage may be into the pleura or even into the great vessels.

Sometimes old connective tissue scars are found in a fresh tear.

The rupture of a papillary muscle or chorda tendinea may give rise to sudden insufficiency of the valve.

Symptoms.—Death from rupture of the heart has suddenly and unexpectedly overtaken persons previously believed to be well. They are found dead in bed or they fall lifeless to the ground, quietly or with a cry.

In other cases the fatal result follows after a few hours. The patient sometimes cries out that something has given way in his heart. He is overpowered by excruciating pain, and the countenance shows great anxiety. The face is pale and the skin cool, and both are covered with a clammy sweat. The pulse is very small and rapid. Very soon there is constant severe vomiting, and sometimes much diarrhœa, so that the appearance is suggestive of cholera. The frequent vomiting has been attributed to anæmia of the brain and to irritation of the vagus. In-

creasing anæmia of the brain shows itself by attacks of fainting, ringing in the ears, and objects becoming black before the eyes. Convulsions may occur just before death.

Physical examination of the heart shows a weak, non-palpable apex beat. The cardiac sounds are very feeble; sometimes, however, the cardiac contractions are lively and irregular. Of great weight in diagnosis is the increasing area of cardiac flatness due to the effusion of blood.

Death usually follows within a few hours; rarely the condition extends over several days. If the hemorrhage is sudden and profuse, the cardiac movements are very much hampered and death occurs from cardiac paralysis. In many cases of heart rupture death comes from cerebral anæmia. There are other cases in which the hemorrhage is too small to impede the movements of the heart or to cause great anæmia of the brain, and death in these instances is attributed to disturbed innervation—so-called shock.

Partial rupture of the heart may be diagnosticated when it affects the papillary muscles or has led to loosening of the chordæ tendineæ, for it then gives rise to the sudden occurrence of insufficiency of the auriculo-ventricular valves. In many cases its onset is accompanied by excruciating pain in the cardiac region, and death may follow quickly. In other cases a compensation of the circulatory disturbance is possible through consecutive dilatation and hypertrophy of the heart.

Diagnosis.—The diagnosis of rupture of the heart can seldom be made with certainty. In complete rupture it is only possible when there are at the same time signs of internal hemorrhage, with increasing cardiac weakness and a rapidly enlarging area of cardiac flatness. In many cases one must be content with a probable diagnosis.

Prognosis.—The prognosis is unfavorable. It is doubtful if healing ever follows rupture.

Treatment.—In persons believed to be the subjects of fatty heart precautionary measures are to be observed, such as the avoidance of all physical and mental over-exertion.

If we think rupture has occurred, we may give a subcutaneous injection of 2 or more grains of ergotin and lay an ice bag over the heart in the hope of stopping further hemorrhage if possible. Wine, ether, camphor, musk, and similar medicaments have been advised to support the failing strength, and large doses of digitalis have been given in the hope of doing good by slowing the heart's action. Morphine is indispensable for the relief of pain, and should be given subcutaneously in full doses.

ACUTE INFLAMMATION OF THE CARDIAC MUSCLE; MYOCARDITIS ACUTA (ACUTE MYOCARDITIS).

According to the extent of the inflammatory process a diffuse and a circumscribed myocarditis are distinguished. If the muscular fibres are chiefly diseased, we speak of a parenchymatous inflammation, while

if the inflammatory process occurs in the interstitial tissue, it is called interstitial myocarditis. Transition forms are quite frequent, as might be expected.

In acute diffuse parenchymatous myocarditis the changes called by Virchow "cloudy swelling" are found in the cardiac muscle. The muscle appears somewhat swollen, has a dull lustre or bacon-like appearance, and is of tender, brittle consistency. On microscopic examination the individual muscular fibres are swollen. They have lost their transverse striation, and their interior is filled with numerous fine closely packed granules. If acetic acid or dilute caustic potash is added to the microscopic preparation, the granules disappear and become a perfectly transparent mass, thereby showing their albuminous nature. At the same time there is seen to be an increase of the muscle nuclei.

If the disease is quite marked and has lasted some time, the process does not remain in the stage of cloudy swelling, but the granules change to fat drops, which no longer dissolve on addition of acetic acid or caustic potash, but assume a gray or black color with osmic acid. The muscle nuclei participate in the fatty degeneration, and if the latter is marked the muscular fibres appear as cylinders thickly filled with large fat drops, and nuclei can no longer be shown in them by means of staining fluids.

Acute diffuse parenchymatous myocarditis is most frequently met with in the ordinary febrile infectious diseases, also after gonorrhœa and influenza, the infection rather than the fever being the cause. The intensity and duration of the infective process, together with the individual resistance, determine which cases are to be exempt from myocarditis. In myocarditis the function of the heart is necessarily impaired, and it may even terminate in cardiac paralysis, which is the chief danger in many infective diseases.

Acute diffuse insterstitial myocarditis also depends almost always on infectious diseases. Leyden has shown that in pharyngeal diphtheria a considerable collection of round cells occurred in the intermuscular connective tissue, which in places gave rise to disappearance of the muscle fibres.

Acute circumscribed myocarditis is usually purulent, and probably in most cases depends on embolism of the coronary artery or its branches. Ulcerative endocarditis, pyæmia, puerperal fever, malignant pustule, glanders, acute articular rheumatism, diphtheria, typhoid fever, and purulent or gangrenous processes in the lungs have caused it.

If the embolic cardiac abscesses depend on ulcerative endocarditis, they are multiple and sometimes in very great numbers. Their appearance changes with their age. The newest ones represent fine gray or grayish yellow points or lines, which under the microscope show little else than an embolus inside a vessel, and on staining with aniline colors they are seen to be filled with low organisms. In older foci they are surrounded by a hemorrhagic zone, and in still older ones suppuration has begun in the neighborhood. The greater the abscess the less is the number of low organisms. The size of the cardiac abscesses varies from that of a pinhead to a pea or even a dove's egg. Sometimes several abscesses lie close together, or they are connected and on section form irregular cavities. Resorption of small abscesses may perhaps be possible, leaving cicatrices behind. In larger abscesses absorption is not

to be expected. In some cases the pus thickens and becomes changed to a mortar-like mass, sometimes with deposit of lime salts. Usually a capsule of connective tissue is formed round the pus and it becomes encysted. In other cases the pus perforates, either into the pericardium, producing diffuse pericarditis followed by death, or into the heart, causing small emboli in the spleen, kidneys, occasionally the brain and skin if the perforation has been into the left ventricle. The emboli are seated in the lung if the right ventricle has been perforated. After perforation into the heart blood is forced into the abscess cavity, and washes it out, and distends the layers of muscle, forming an acute cardiac aneurysm. The latter may rupture into the pericardium and produce death from internal hemorrhage and cardiac paralysis. Cure of cardiac aneurysm and the formation of a protective connective tissue membrane inside does not seem to occur, probably because the continuous blood current interferes with the reparative process.

Sometimes the formation of a long fistulous suppurating tract precedes the perforation of a cardiac abscess, so that the point of rupture is quite distant from the location of the abscess. Thus it may happen that the pus forces its way between the two endocardial layers of a valve to its tip, and then escapes into the ventricle.

CHRONIC INFLAMMATION OF THE CARDIAC MUSCLE; MYOCARDITIS CHRONICA (CHRONIC MYOCARDITIS).

DEFINITION.—A chronic disease of the heart muscle or interstitial tissue, leading usually to cardiac weakness and death.

ETIOLOGY.—Among the causes of chronic myocarditis the following are mentioned: the effects of cold; injuries, as a fall or blow on the chest; and physical over-exertion, the so-called rheumatic-traumatic myocarditis.

Frequently the infectious diseases, as acute articular rheumatism, malaria, syphilis, give rise to infectious myocarditis. Chronic muscular rheumatism is said by some to predispose to chronic myocarditis.

There is a toxic form of myocarditis depending on the abuse of alcoholic drinks, tobacco, and lead-poisoning.

The cases depending on changes in the coronary arteries are called vascular myocarditis, and to this same class belong cases depending on metabolic diseases, as gout and diabetes mellitus.

Chronic myocarditis not infrequently accompanies chronic nephritis, especially the sclerotic form, and is called nephritic myocarditis. It is also found in cases of long continued passive congestion.

In quite a number of cases it may be regarded as a senile change, especially when it occurs in conjunction with endarteritis of the coronary arteries—senile myocarditis.

Sometimes the myocardium suffers a chronic change from the beginning; sometimes acute changes have preceded, which become chronic; lastly, chronic myocarditis may depend on a preceding peri- or endocarditis.

Chronic myocarditis is unquestionably more common in men than in women, as its causes would indicate, and it occurs most frequently after the fortieth year of life.

Pathological Anatomy.—The peculiarity of a chronic inflammation of the cardiac muscle consists in its tendency to the formation of connective tissue plates or callosities; whence the name myocarditis interstitialis sclerosa.

In examining the heart after death in such a case the section of the cardiac muscle should be made parallel to the course of the fibres in horizontal lines, as the extent of the change can only be made evident in this manner.

Cardiac callosities appear at first as grayish red, later gray white spots, lines, or plates which pervade the heart muscle more or less abundantly. Sometimes they appear as pale yellow or brownish spots, which on microscopic examination consist of the remains of contracted and fatty muscular fibres and coloring matter.

The extent of these callosities is very various. Sometimes they appear to be simply an increase of the intermuscular connective tissue, while in other cases they form branched and extensive plates 3 or 4 centimetres broad. Again, they are dense solid nodules. Sometimes they extend through the whole thickness of the heart-muscle, so that pericardium and endocardium are separated by connective tissue only. They may be so numerous that the greater part of the cardiac muscle is replaced by them.

They are found most frequently in the wall of the left ventricle, especially near the apex, and next in frequency in the septum of the ventricles. On the right side of the heart they occur for the most part only during fœtal life, and then often give rise to congenital heart disease.

If the cardiac muscle contains connective tissue callosities of small number and extent, they may be without importance, and only be found accidentally at autopsy. If, on the other hand, they be in large number and extensive, they give rise to insufficiency of cardiac power, manifested by cardiac weakness and evidences of passive congestion, just as in uncompensated valvular lesions.

In addition to the appearances of chronic myocarditis there are usually other changes in the heart. The epicardium is frequently cloudy and thickened, and the same thing may be found on the endocardium. Occasionally the heart muscle is hypertrophied in places unaffected by the disease.

Frequently the myocarditis depends on disease of the coronary arteries, the intima being thickened and narrowed in places. Sometimes the process is a thrombotic closure of a branch leading to a white or hemorrhagic infarction of the muscle, necrotic softening, absorption, and formation of cicatrices.

Sometimes the papillary muscles are the seat of the process. They shrink and change at their apices to stiff tendinous bands. Their function is thereby impaired, and in connection with these changes there may be shrinkage of the valves leading to insufficiency.

As a result of chronic myocarditis the condition called true cardiac stenosis may follow. The connective tissue forms a ring at the point

where the pulmonary artery is given off, and contracts so as to cause considerable stenosis at the beginning of the pulmonary artery. Such changes may occur during fœtal life, but may also come on later. The same thing has been found at the left conus arteriosus, giving the appearances of stenosis of the aortic orifice.

Chronic cardiac aneurysm may result from chronic myocarditis. Since the cicatrices are not capable of contraction, they gradually yield to the blood pressure inside the heart, and form a sac with perhaps a narrow, shrunken entrance. These saccular dilatations, known as chronic aneurysms, are most frequently found in the left ventricle near the heart's apex. Of 87 cases collected by Pelvet, 55 were in this locality, and only 3 in the right ventricle. Sometimes there are several aneurysms in one heart, which usually lie close together. The size of the aneurysms may equal that of the heart, or even surpass it, as was seen in a case by the writer some years ago. If the cardiac aneurysm has reached a certain size, all traces of muscular tissue disappear from its wall, and there may be calcification in places. Thrombi may be formed in concentric layers in it. Very frequently there are adhesions between the outer layers of the aneurysm and the parietal layer of the pericardium, so that the aneurysm projects into the left pleural sac or is largely surrounded by the left lung. If the aneurysms are in the septum ventriculorum, they extend almost without exception into the right side of the heart on account of the greater blood tension in the left side.

Symptoms.—Symptoms may be wanting in chronic myocarditis if the connective tissue callosities do not interfere with the working power of the cardiac muscle or if the nerves are not implicated—latent chronic myocarditis. Sometimes the diagnosis is rendered very difficult from the ambiguous character of the symptoms. Generally the symptoms show the cardiac power to be more and more insufficient. The patient complains of palpitation and shortness of breath on slight movements of the body, and is able to do but little. Pain in the cardiac region comes on with the palpitation, and streams into the left arm or downward into the epigastrium. Apex beat, cardiac impulse, and cardiac sounds are very weak. Sometimes, instead of the first sound, there is a systolic murmur. The gallop rhythm is found either sometimes or in advanced stages is constant. The cardiac movements are arrhythmical. Very often the pulse is intermittent, at times prolonged. The sphygmographic tracing shows that the pulse beats are of unequal power and duration. The patient is liable to have catarrh of the air passages and a marked fulness of the cervical veins. He is usually cyanotic, and there is very apt to be disturbance of the digestion. If passive congestion gets the upper hand, œdema of the skin and serous cavities comes on, enlargement of the liver, bronchial catarrh, and hemorrhagic infarction; and if the cardiac strength cannot be increased, death follows from œdema of the lungs, inflammation of the same, hæmoptysis, or cerebral hyperæmia. Sudden death may occur, the cause of which is not explained by autopsy.

The recognition of a chronic cardiac aneurysm is very difficult during life, and can seldom be carried farther than a probability.

Prognosis.—The prognosis of chronic inflammation of the cardiac muscle is always grave. So long as diuresis can be maintained the case

continues fairly well. If the trouble depends on syphilis, therapeutic measures directed to this may be of service.

TREATMENT.—Absolute rest of body and mind is essential, and an easily digested and nourishing diet is of prime importance. Ice bags over the præcordia may be used in rapid and irregular cardiac action. If sudden cardiac weakness or insufficiency occur, the case is to be treated like uncompensated valvular lesions. Energetic antisyphilitic treatment is in order in appropriate cases.

THE FAT HEART (COR ADIPOSUM).

SYNONYMS.—Adipositas seu Lipomatosis seu Obesitas cordis; Lipoma capsulare cordis; Atrophia cordis lipomatosa; Fat infiltration of the heart.

DEFINITION.—Accumulation of fat about the surface of the heart or between the muscular fibres, or both.

PATHOLOGICAL ANATOMY.—In the healthy heart fat is always found in moderate quantity in the subepicardial connective tissue, especially in the grooves and about the great vessels, at the lower edge of the right ventricle, and near the apex. Excessive increase of the same leads to fat heart. In this condition the masses of fat are found increased, not only in the places mentioned, but they also extend over the surfaces of the ventricles. At first the right ventricle is surrounded by a thick layer of fat; later and in advanced disease the left ventricle also. Lastly, the entire heart is covered with a thick capsule of fat which may attain a diameter of more than one centimetre. It is not uncommon to find the fat of a deep yellow color, like sulphur; in other cases it is pale yellow.

On section it is frequently seen that the growth of fat is not confined to the subepicardial connective tissue alone, but has forced its way deep into the heart muscle along the intermuscular bands of connective tissue. In many cases the muscular tissue of the heart is extremely thin, and of brownish yellow or fawn color. Such a heart is especially liable to rupture. Not infrequently endarteritis is seen in the coronary arteries and aorta.

ETIOLOGY.—Among the causes of fat heart the chief one is general obesity. It is especially observed in persons who unduly enjoy the pleasures of the table and take little exercise. A diet rich in the carbohydrates is held responsible in some cases. It is well known that the consumption of beer, wine, and alcoholic liquors tends to obesity. The fat heart is never found in infancy; it occurs only after maturity, and the tendency to it increases as the age advances. It is usually seen in persons who have passed the fortieth year, but cases are on record as young as sixteen, most of them already confirmed inebriates. It occurs most frequently in males, though women who suffer from amenorrhœa or sterility, and some after childbed and the climacteric, may have it. The disease seems to be hereditary; it certainly runs in families. It is

more marked in some races and localities. A few cases follow loss of blood or other fluids and anæmic or cachectic conditions.

SYMPTOMS.—Cases are not rare in which well marked fat heart is accompanied by no symptoms, and is discovered accidentally at autopsy. This is the so-called latent fat heart.

In other cases there may be no striking symptoms, but, nevertheless, the disease gives rise to sudden death from spontaneous rupture of the heart, due to the diminished resistance of the cardiac muscle.

In a third group of cases there are the symptoms of insufficient cardiac power, which may come on suddenly after great exertion. Overdistention of the venous system is observed, and slight filling of the arteries. There are feebleness or loss of the apex beat and a diffuse cardiac impulse. Percussion often shows increase of the cardiac dulness from dilatation. The cardiac sounds are feeble, and not rarely there is a systolic murmur instead of the first sound. In many cases one hears the gallop rhythm. The cervical veins usually are very full, and sometimes show a venous pulsation. The radial pulse is abnormally small as a rule, and is frequently arrhythmic. The peripheral arteries may show calcification and arterio-sclerosis. There are frequent attacks of palpitation, either occurring spontaneously or after slight physical or mental excitation. These may be accompanied by pain in the præcordia, which frequently also extends down the left arm. In many cases there is a small pulse, abundant perspiration, and subnormal temperature. Dyspnœa is frequent, with occasional asthmatic attacks, called by some authors cardiac asthma. Some writers call attention to three symptoms as of diagnostic importance—namely, slow pulse, pseudo-apoplectic seizures, and Cheyne-Stokes respiration.

The retardation of the pulse may be very great. It has been reported as being only forty, thirty, fourteen, or even eight beats to the minute. This abnormally slow pulse may last for a long or a short time. Its cause is irritation of the vaso-motor nerve centres, and it may follow, cerebral anæmia.

The pseudo-apoplectic attacks resemble apoplexy, as their name indicates. The patient in many cases suddenly loses consciousness and falls down. On awaking he rapidly regains consciousness. Sometimes weakness of one of the extremities remains for a short time; more infrequently there is permanent paralysis of one side, which of course cannot depend on cerebral anæmia, but is attributable to cerebral hemorrhage.

The number and duration of these attacks vary greatly. In many patients weeks and even months pass before such attacks are repeated; in others, on the other hand, they are repeated several times in the course of a few days. The quicker the attacks follow each other the shorter is their duration, as a rule. Sometimes the patient is made aware of their approach by a retardation of the pulse or other peculiar premonitory sensation, so that it may be warded off. Stokes reports a case where the patient assumed the knee-elbow position and lowered the head, thus aborting the attack.

Sometimes twitching of the extremities occurs during the attack. Almost always there are alterations of the pulse and disturbances of the respiration.

Cheyne-Stokes respirations are readily recognized. They become

deeper and deeper, take on the character of dyspnœa, become sighing and stertorous, and gradually increase to apnœa. Leube says the pupils are contracted during the apnœa, and Traube observed twitching of some of the muscles sometimes toward the end of the respiratory pauses. Fräntzel gives the duration of apnœa as up to forty seconds.

Sometimes Cheyne-Stokes respiration occurs only during sleep; in other cases sleep or disturbances of consciousness come on during the respiratory pauses. Fräntzel found that this form of breathing sometimes came on from the use of narcotics, as was also observed by Eichhorst. Not infrequently these appearances are observed during the pseudo-apoplectic attack. At times it is only a temporary phenomenon, but it may drag on for weeks or even months.

The duration of the complaints resulting from fat heart may extend over several years, with alternate improvement and relapse.

The fatal issue is frequently sudden. It may follow from exhaustion of the heart, and cardiac paralysis from over-distention, or more rarely from rupture. Such an unfortunate termination has been observed after childbirth. In other cases death occurs quite unexpectedly from cerebral hemorrhage. Pseudo-apoplectic attacks under some circumstances may directly lead to death. Sometimes acute œdema of the lungs comes on, the left side of the heart being paralyzed and the right continuing to act. Embolism of the pulmonary artery and hemorrhagic infarction have been observed as causes of death.

In many cases repeated copious epistaxis occurs, which reduces the strength and hastens the fatal issue. Increasing insufficiency of the cardiac power, already described, brings in its train œdema of the skin and serous cavities, hepatic engorgement, diminution of the amount of urine, albuminuria, bronchial catarrh, hemorrhagic infarction, inflammation of the lungs, pulmonary œdema, somnolence, convulsions, and so forth.

Diagnosis.—The diagnosis of fat heart does not reach beyond a probability, as a rule. Great retardation of the pulse, pseudo-apoplectic attacks, and Cheyne-Stokes respiration do not often occur at the same time, and but little can be determined from these symptoms. If insufficiency of the heart comes on first of all, myocarditis must be excluded, and the history and etiology, together with existing signs of general obesity, must be taken into account.

Prognosis.—Fat heart presents an unfavorable prognosis, for the patient comes under treatment so late in the disease usually that energetic treatment is not advisable nor is a permanent result likely to follow. Many patients are unable to give up their bad habits, or with the slightest improvement they return to them. An irregular pulse is said to give an unfavorable prognosis.

Treatment.—In the treatment of fat heart we must distinguish between the plethoric and the anæmic forms. In both prophylactic measures are in order, though usually the patient first comes under the hands of the physician when such measures are of no avail.

In the plethoric form the general treatment is that for obesity, the amount of food being limited, as well as of the drink. Carbohydrates are to be excluded as far as possible, but fats may be allowed. Systematic exercise, particularly mountain-climbing in moderation, is to be

advised. Iodide of potassium in five-grain doses or more three times a day has been recommended. Good results have followed the water cure at Marienbad, Kissingen, Homburg, Wiesbaden, Karlsbad, and other places.

In anæmic fat heart all foods are to be forbidden which lead to the accumulation of fat, especially carbohydrates, and the quantity of fluid allowed is to be kept at the lowest possible limit, but wine may be administered as needed. The patient should spend much time in the open air, but should walk neither much nor far, so that the heart shall not be overtaxed nor tired. Mild chalybeates should be ordered, and in the summer season mild laxative iron mineral waters may be used to advantage.

If insufficiency of the heart occurs, digitalis or its substitutes should be ordered, as in uncompensated valvular lesions. With increasing evidences of passive congestion, diuretics, drastic cathartics, or diaphoretics should be added.

For symptoms of cerebral anæmia the head should be kept low and camphor, musk, or ether given internally, while the temples may be rubbed with various stimulants to advantage.

Asthmatic attacks are sometimes best combated by causing the patient to lie down in a dark chamber with an ice bag over the præcordia, and giving small pieces of ice to hold in the mouth, or water-ices in teaspoonful doses, or perhaps a little strong black coffee. Morphine or other narcotics should be used with caution, since they may readily, as mentioned above, bring on Cheyne-Stokes respiration and cerebral anæmia. Germain Sée extols the inhalation of pyridia. Tincture of strophanthus and sparteine sulphate have been used with good results. If the threatening symptoms have been allayed, small doses of digitalis, kept up for a long time, are strongly advised.

CARDIAC THROMBOSIS AND ANEURYSM; MORBID GROWTHS AND PARASITES.

By CHARLES E. QUIMBY, M. D.

CARDIAC THROMBOSIS.

Definition.—Under a strict interpretation of the term cardiac thrombosis must be held to include all *ante-mortem* formations of coagula within the heart, although those clots which develop just preceding death, simply as the result of vascular stagnation, possess no clinical significance even when they are the immediate cause of arrested heart action, since they are primarily dependent upon precedent and predominant pathological conditions, and only hasten, but do not determine, the final result.

Clinically, cardiac thrombi appear in two classes, as they are developed slowly or rapidly. In the more acute forms, when the process is that of coagulation, they are usually termed heart clots or thrombi; when of slower growth from gradual fibrinous deposit, and especially if they project into the heart cavities, they are described as cardiac polypi; while the smaller fibrinous deposits that develop on protuberant, roughened, or eroded points on the heart walls or valves are more commonly called vegetations.

Etiology.—Three factors are involved, singly or in combination and with varying ratios, in the production of cardiac thrombi. They are (1) retardation of the blood current; (2) obstructions to the blood current and changes, both anatomical and pathological, in the endocardial surfaces; and (3) modifications of the blood elements. The primary causes of cardiac thrombosis are found in corresponding classes.

In the first, weakened heart action, either with or without dilatation, is the most frequent cause. When with this is associated some obstruction to the circulation, as in lobar pneumonia, the conditions are most favorable for the formation of acute cardiac thrombi in the right ventricle and pulmonary artery. They may form as well in the left ventricle under similar circumstances, which, however, are less common in connection with the general circulation. Such thrombi are more frequent in conditions of extreme systemic depression and exhaustion, and have been called "marantic thrombi." These conditions are found especially with fatty and fibroid degeneration, chronic inflammation of the myocardium, pericardial adhesions or effusions, plastic pericarditis, and cardiac atrophy of old age. They are not infrequent also in the later stages of the infectious diseases, and particularly such as run a prolonged course or are characterized by a peculiarly virulent toxin,

as typhoid for the first and diphtheria for the last, and typhus as combining the two qualities.

In addition to these mechanical causes, pathological processes in the endocardium play a prominent part in determining thrombosis, although it may often be difficult to define their exact relations. Omitting all discussion of any assumed vital action of the endothelial cells, it is certain that erosions and ulcerations of the endocardium favor both coagulation and fibrin deposits, which, though small at first, may be the starting point of large thrombi. Indeed, this is probably the more usual origin of the chronic fibrinous cardiac polypi. Such thrombi as form on roughened valvular surfaces in acute or chronic endocarditis less frequently develop sufficiently to give the common symptoms of cardiac thrombosis, but are none the less important as the source of emboli.

The initiation of thrombosis is frequently at some point where there is a natural obstruction or anatomical retardation of the blood current, as among the columnæ carneæ and in the appendices auriculorum. Cardiac aneurysms thus frequently, and valvular aneurysms occasionally, become the starting points for cardiac thrombi. In the former case they are apt to form slowly, to be composed of laminated fibrin, and to develop into true cardiac polypi. A valvular stenosis also, more especially at the mitral orifice, may attain such a degree as to determine the starting point of thrombosis when it is favored by other causes. It has been claimed that small clots from the veins floating as emboli may find lodgement on the cardiac walls and become the exciting cause of further coagulation. Such cases must be extremely infrequent. The etiological relations of changes in the blood itself to thrombosis in general are still somewhat indefinite, although the statement is generally made, in connection with most of the specific diseases at least, that the blood shows a diminution of coagulability. Yet the relative frequency with which thrombosis is associated with infectious processes forces recognition of blood changes as at least clinical predisposing causes of cardiac thrombosis.

Pathology.—The differentiation at autopsy between true cardiac thrombi and post-mortem, or even immediately ante-mortem, clots is usually not difficult. When recent clots have been formed rapidly, either post-mortem or just at the time of death, they may retain the dark color of the blood, but more frequently from slower coagulation they are either in large degree devoid of blood coloring matter, or perhaps stained more distinctly in their lower layers and along the borders. Such clots are of a dirty straw color, at times somewhat translucent, and usually soft and œdematous. They are of gelatinous consistence, and often bear on their surface the impress of the valves and columnæ carneæ. Their recent origin is also shown by their direct continuity with clots of similar characteristics extending widely in the veins. When lifted from their seat they are easily separated from the cardiac wall without rupture, although they are readily torn apart.

True cardiac polypi and the older thrombi are formed by gradual deposition of fibrin upon some roughened or projecting point or at a point where the blood current is detained. They are thus more frequently found at the apex of the ventricles, particularly the left, and in

the auricular appendices. As the successive layers are formed varying amounts of coloring matter become entangled in the fibrin, so that their laminated structure is accentuated by differences in coloring. The more recent layers are light in color, but gradual shrinking and condensation concentrates the pigment until some layers are made quite dark. On the surface these thrombi are of a dull grayish white color, and may present a ragged, irregular outline and roughened surface, although usually smooth and frequently rounded in contour (*végétations globuleuses*). They are firmly attached to the cardiac wall, commonly by a broad face, but they may develop with a stem in true polypoid form, and hang free in the cardiac cavities or even extend through a valvular opening from one cavity to another. In size they vary from the smallest nodule to a mass which at autopsy seems to completely fill the auricle or ventricle. In shape they may be broad-based cones, rounded or flattened ovoids, or drawn out into long cords that extend into the vessels. Although firmly attached, the mass is usually dry and friable, and liable to be torn in efforts to remove it. Large chronic thrombi usually undergo central degeneration and frequently become cystic. The fluid contents is composed principally of disintegrated fibrin elements with some cells and leucocytes, but not in such numbers or form as to justify the name of pus cysts which has sometimes been applied to these puriform thrombi.

Distinct organization of cardiac thrombi has been observed, but is even more infrequent than the central deposition of calcareous salts and the formation of firm concretions, which happens but rarely.

Symptoms.—The associated symptoms indicating cardiac thrombosis will vary within wide limits and depend on the rapidity of development as well as the location of the thrombus. When heart clot occurs in connection with pneumonia or in the later stages of chronic pulmonary disease, the increase of dyspnœa, cyanosis, and venous turgescence, together with a suddenly disturbed and weak heart action, may be sufficiently acute to attract attention to the heart; but not infrequently these symptoms develop more slowly, and appear to be only an aggravation of similar conditions previously existing. When sudden or rapid advent of such symptoms is attended by diminution in frequency of the heart action, or rarely by violently irregular beating, and a systolic murmur develops in the right heart, with loss of the tricuspid valvular sound, a diagnosis of thrombosis may safely be made. In the most acute cases, when primary and extensive thrombosis occurs in a weakened and dilated heart, or when acute extension follows an old thrombosis, a few gasping respirations and sudden deep cyanosis are the only warnings before the succeeding coma has ended in death. If the finger happens to be on the pulse at the time, a few fluttering beats will be felt interspersed with heavy pounding throbs.

In the more chronic forms of thrombosis the symptoms are less definite and much more variable, and in many instances cannot be ascribed to this condition with any certainty. Such thrombi usually cause no subjective symptoms during their earlier development or until they begin to interfere with the heart action. The opportunity of watching the development of the physical signs is thus lost.

When patients first make complaint of their heart, its action will

be found disturbed in both rhythm and force, as indicated by intermittence, irregularity, and unevenness of the pulse. Auscultation at this time will probably reveal a systolic or possibly diastolic murmur, but without distinctive characters, unless it can be located in the right heart, where thrombosis is most frequent. As the growth begins to obstruct more decidedly the blood current, the disturbance of heart action increases, particularly under excitement or exertion, and is attended by progressive dyspnœa, cyanosis, or pallor and surface coldness. This obstruction, if prolonged, may induce œdema of the feet or even general anasarca.

It seems unnecessary to enumerate all the unique symptoms possibly developed by pressure, such as a systolic murmur in the pulmonary artery due to compression by a distended left auricle, since they are not specially significant of thrombosis, and at the best are extremely rare. Moreover, they may all be determined from the anatomy of the parts. When firm fibrinous vegetations form on roughened valves they are usually small in size, and give rise to few symptoms unless torn from their attachments and converted into emboli. If sufficiently large to obstruct a valvular opening or interfere with valvular action, the first symptoms will be objective, but the resulting valvular murmur cannot in most instances be distinguished with any certainty from similar murmurs of different origin. It is only when new murmurs are found to have developed or old ones are suddenly increased, without attendant signs of acute inflammation, that auscultation affords aid in diagnosis. When such murmurs are in the right heart they are still more suggestive of thrombosis. In a few cases inspection may show jugular pulsation, and when an auricle is markedly distended corresponding increase of cardiac dulness may be detected.

Complications and Sequelæ.—When cardiac thrombi do not cause immediate death the most important complications are cerebral embolism and hemorrhagic pulmonary infarction, both of which are associated with thrombosis in the left heart. The sequelæ of right cardiac thrombosis are largely the result of impeded circulation. Small embolic pulmonary infarctions or an embolic pneumonia may also occur.

Diagnosis.—Slowly developing thrombosis cannot be recognized with certainty. Its probable existence must be determined by exclusion of other more frequent causes for progressive cardiac disturbance with or without murmurs. In acute thrombosis the evidences of severe systemic shock and cardiac obstruction in connection with a suddenly developed systolic murmur must form the basis of diagnosis. Rupture or perforation of a valve and rupture of a chorda tendinea in ulcerative endocarditis are the two conditions most closely simulating thrombosis. In the case of rupture the resulting murmur is either regurgitant or double. It is quite certainly obstructive with thrombosis. In rupture the lesion is usually in the left heart, and the cyanosis much less marked in proportion to the dyspnœa. Acute thrombosis is most frequent in the right heart and the venous turgescence is prominent. In rupture of the heart, which may also be mistaken for thrombosis, systemic and cardiac shock will be equally prominent symptoms with dyspnœa and cyanosis, but the extravasation of blood into the pericardium will give

lateral increase of cardiac dulness and indistinct apex beat. The murmurs of thrombosis will be absent.

Prognosis.—That cardiac thrombi may exist for a considerable time without causing symptoms, and that in some instances they grow with great slowness, does not prevent the prognosis from being extremely unfavorable. Small coagula may become organized, and occasionally a thrombus that starts in an aneurysmal sac may simply fill the cavity and not extend into the heart; but the vast majority of cases are surely progressive, while acute thrombi of large size prove fatal within a period of a week at the latest. Reported cases of recovery from acute cardiac thrombosis must excite grave suspicions of error in diagnosis.

Treatment.—Little can be done directly to prevent or arrest the thrombosis. The use of the alkaline carbonates, more particularly the carbonate of ammonia, and later the aromatic spirits of ammonia, has been recommended and largely employed under the belief that they diminished the tendency to coagulation. Such use must be based principally upon theoretical grounds, as the clinical evidence of their value can never be very definite. The ammonia preparations nevertheless afford a satisfactory and usually available cardiac stimulant, and, as there is certainly no evidence that they tend to increase the formation of thrombi, their use in this condition is to be commended and to be guided by their action upon the heart.

Absolute quiet of body and mind must be enforced in all cases of acute thrombosis. Pain and restlessness may be relieved by the cautious use of opium. Dyspnœa is best treated by persistent inhalation of oxygen in small amount and the use of moderate doses of glonoin. If cardiac stimulants other than ammonia are required, strophanthus and caffeine are to be preferred to digitalis. When extreme cyanosis is followed by irregular heart action, strychnine will afford some relief, and irregular respirations may similarly be assisted for a time by small doses of atropine. In cases where a slowly developing thrombus or cardiac polypus is believed to exist, little can be done beyond avoiding all causes of increased heart action by which the danger of rupture of the thrombus and the formation of emboli would be augmented.

Nothing is to be hoped for from attempts to cause absorption by the use of mercury and the iodides. Such treatment will rather tend to increase the fibrinous deposit.

CARDIAC ANEURYSMS.

Definition.—The term cardiac aneurysm, which at one time was applied to all forms of dilatation of the heart without hypertrophy, is now restricted to limited depressions and cavities formed by localized stretching of the cardiac walls and to diverticula within the walls or septa which communicate with the heart cavity. According to the rapidity and manner of their formation they are classified as acute and chronic. Following the accepted classification of arterial aneurysms,

those which are formed by rupture of the endocardium and extravasation of blood, and which correspond very closely with the acute, may be termed false aneurysms, while such as are developed by stretching of the cardiac tissue are the true cardiac aneurysms.

Etiology.—Decreased resistance and increased vascular tension are the two mechanical factors which either singly or in unison underlie every cardiac aneurysm. Any condition producing either thus becomes etiological. Age, as might be expected, is a prominent predisposing cause through its natural tendency to excite degenerative changes in all the tissues. The condition has been found, however, in young children. It is also much more frequent in the male sex as a result of the high arterial tension attendant upon all forms of muscular exertion. The ratio is as high as three to one. It is less easy to determine the relative frequency of the acute and chronic forms, as doubtless in some instances an aneurysm which is started by a rupture or ulceration of the endocardium may become chronic, and the succeeding pathological changes render a determination of its exact cause impossible. By far the larger proportion are of the chronic form. The direct exciting causes are increase in cardiac vascular tension and localized weakening at some point in the cardiac wall. It is impossible to state definitely whether the first of these alone ever produces aneurysm in a previously healthy heart. It is known that such strain has caused complete rupture of the heart. There is, therefore, every reason to suppose that a partial rupture thus produced may be the starting point of an aneurysm, and to assume that the degenerative changes found post-mortem were secondary rather than primary. With primary changes weakening the cardiac wall, increased tension becomes a prominent factor in determining the development and growth of an aneurysm.

The pathological processes tending to the formation of acute aneurysm are all degenerative in character. Acute endocarditis may lead to softening or even ulceration of the endocardium, which is followed by rupture and extravasation of blood between the muscular layers of the heart or fibrous layers of the valves. Aneurysms from ulceration are more frequently found in the valves and interventricular septum. Similarly, acute inflammation of the myocardium may result in softening and partial absorption at some point in the cardiac wall. The overlying endocardium becomes depressed and an aneurysmal cavity is started, which later may be more rapidly enlarged by rupture of the endocardium and inclusion of the softened area. Or, again, the myocarditis passes on to suppuration, and an abscess is formed which eventually communicates by rupture with a heart cavity. It is also claimed that in many cases of myocarditis there is simple extravasation of blood into the softened area (cardiac apoplexy), and that later a communication is established with the heart cavities without suppuration after the clot has undergone cystic transformation.

Similar conditions are the result of non-inflammatory processes. Fatty degeneration, syphilitic or tubercular new growths, may thus each be the starting point for a focus of softening that will develop into an aneurysm by the processes just described, or by simple distention of the cardiac cavity and localized stretching of the wall without rupture. The development of chronic aneurysm is almost invariably by gradual

stretching of some weakened area in the heart wall, which still retains a good degree of strength if not resiliency. Such areas are largely fibroid. All the causes of local cardiac fibrosis thus become etiological to cardiac aneurysm. Among these, obstructions to the cardiac circulation from atheroma and embolism of the arteries are most prominent. It is stated that fibroid tissue in the heart shows less tendency to contract than similar tissue elsewhere. The mechanical conditions under which it is developed, and by which it is put to constant strain before becoming fully organized, seem better to account for the fact that these cardiac fibroid plates so frequently become stretched instead of contracting, than the assumption that the tissue itself possesses peculiar properties. Since the degree of fibroid repair following muscular degeneration is variable, it is evident that the line between acute and chronic aneurysm cannot be very definite.

PATHOLOGICAL ANATOMY.—By far the larger number of cardiac aneurysms, both acute and chronic, are found at the apex of the left ventricle and usually upon the anterior wall. The ventricular septum, particularly near its upper portion, is their next most frequent seat. The aneurysmal tumors vary in size from that of a pea to that of a cocoanut (Quain), and may be single or multiple. In the latter case they usually lie adjacent to each other, and may communicate with the ventricle by a common or individual opening. When the aneurysm is small or of the dissecting variety, the heart may present no abnormal appearance externally. Usually, however, it is enlarged—at times hypertrophied and again dilated. If the new cavity is large, it may distort the outlines of the heart or appear as a distinct tumor presenting various shapes, either rounded, globular, conical, or fusiform. When arising near the base of the heart it may grow upward about the base of the aorta. Quain reports a case which was an elongated sac winding around the aorta. Pericarditis is quite constant over such aneurysms. It results in adhesions that may be local or extend throughout the pericardial sac.

Viewed from within, cardiac aneurysms present an equally varied anatomy. In some cases the cavity is nothing more than a saucer-shaped depression on the ventricular wall, which offers little or no obstruction to the blood current, and affords no opportunity for fibroid deposit or clotting. In others, with the opening into the heart still the largest part of the aneurysm, it may appear like a toy cup with flaring brim set into the heart wall. Less frequently, when the aneurysm starts from a point of endocardial ulceration, the blood may make its way between the muscular fibres in a canal of rather uniform size, and possibly form eventually a second opening communicating with the same or another heart cavity.

Most commonly, and particularly in chronic cases, the tumor presents a more or less definitely constricted neck, which connects the sac with the heart cavity through an opening of any size from the most minute to one of several inches in diameter. When this "mouth" is sharply outlined it indicates usually an equally sharp limitation of the primary degeneration and the persistence of good nutrition in the surrounding tissue. In such cases, therefore, the development of fibrous tissue at this point is more prominent, so that the opening and possibly

part of the neck are firm and thicker than the rest of the sac, being at times almost cartilaginous in consistency. The smaller openings are often regular and the surrounding surfaces smooth, while the larger ones are more apt to be irregular and rough. Within the sac the walls are usually smooth when formed of uncovered endocardium or layers of fibrin, but when the aneurysm has developed by rupture the inner surface is often composed of ragged muscular tissue, and in any case irregular deposits of fibrin may line the cavity with a rough, shaggy membrane.

In the earlier stages of chronic cases the sac wall is usually composed of all the heart tissues, with the addition of fibroid elements in varying proportions. As the cavity enlarges the muscular fibres tend to disappear at the point of most rapid growth, leaving only a fibroid membrane between the peri- and endocardium, which latter are generally persistent unless the starting point was in ulceration or rupture. Very rarely these membranes alone form the sac wall. In all cases where the aneurysm approaches the surface of the heart thickening and adhesions of the pericardium reinforce the weakening tissues and tend to delay growth. Occasionally the walls become indurated from excess of fibrous growth or have cartilaginous plates developed in their substance, but usually they become thinned, and quite certainly reach that condition before they are seen at autopsy. They are found thicker than the normal heart wall only when there has been precedent muscular hypertrophy. In the more open-mouthed and shallow aneurysms the blood may remain fluid, but when the cavity becomes sacculated there is a tendency to fibrin deposit and coagulation. The fibrin formation is usually by rather clearly defined layers, of which the deeper ones may undergo a partial organization and become firmly adherent to the sac wall.

Blood clots are quite constantly present in the larger and less open tumors. They are adherent, with the outer portions partially organized. Their surfaces are at times smooth, but often rough and irregular, thus increasing the danger of laceration and the formation of emboli. The very rare recorded cases in which a cure or arrest of the growth had apparently occurred seem to show that such a result is attained rather by thickening and induration of the outer fibrous layers than by filling the sac and obliterating the cavity.

Symptoms.—Cardiac aneurysms, as a rule, present no characteristic or typical indications of their presence. About one fourth of the recorded cases gave no symptoms and were discovered only on autopsy. All the subjective sensations are due to disturbance of heart action, which may be manifest in both rhythm and force. Palpitation and irregularity are perhaps more frequent at first than intermittence. Pain and dyspnœa are later symptoms associated with or followed by paleness and the cyanosis so characteristic of obstructed circulation. Although murmurs may be heard, they present no peculiarity by which their cause can be determined with certainty, except that they simulate ventricular rather than valvular murmurs. Indeed, it is probable that they are generally dependent upon other conditions than the aneurysm. The apex beat is usually diffuse and weak, and may be displaced.

With the larger aneurysms the area of cardiac dulness is appreciably

increased, more frequently downward and to the left; but it is only when an irregular outline of dulness is associated with weak impulse at the protuberant point that it becomes at all significant. A large anterior aneurysm may cause bulging of the thoracic wall and a localized impulse, or even produce absorption of the tissues; but this could happen only with an auricular aneurysm, which could hardly be distinguished from aortic disease. The gradual development of the signs of chronic pericarditis following a prolonged period of disturbed heart action is as suggestive of aneurysm as any symptom can be.

PROGRESS AND TERMINATION.—The majority of cardiac aneurysms are of very gradual development, and their growth is comparatively slow, even when they have attained considerable size. Even such as are of acute origin from ulceration or acute softening follow a similar course when their onset is not fatal. Variations in the rapidity of their growth will depend largely upon the original cause and its relation to cardiac nutrition. Owing to the impossibility of fixing the time of their beginning, it is equally impossible to approximate even an average duration. Still further, intercurrent or precedent heart lesions either hasten or determine the fatal termination. They all tend in one direction. When the aneurysm is the direct cause of death, it is usually by rupture, which may take place into the pericardium or into an adjacent heart cavity. If the walls remain intact, the growth may mechanically so obstruct the heart action as to cause death. Even a small aneurysm burrowing in the muscular wall may produce the same result. This is most common with aneurysms starting near the base of the aortic valves. Valvular aneurysms also may so obstruct the circulation as mechanically to arrest the action of an already weakened heart.

DIAGNOSIS.—Recognition of this condition is always uncertain, and the diagnosis must remain doubtful until confirmed by autopsy. Rarely a large surface aneurysm may cause a suggestive change in the outlines of cardiac dulness.

PROGNOSIS.—Post-mortem evidence that cardiac aneurysms of considerable size may exist without material interference with the circulation mitigates somewhat the severity of what would otherwise be an absolutely unfavorable prognosis from a theoretical standpoint. In all cases, however, where a probable diagnosis is made a fatal termination cannot be far off.

TREATMENT.—Assuming a diagnosis to have been made, the treatment would differ somewhat from that for simple cardiac degeneration. While all the usual measures for improving muscular nutrition, as iron, oxygen, and proteid food, are appropriate, cardiac stimulants and everything tending to increase vascular tension should be avoided or used only under compulsion. Theoretically, the same line of treatment employed in inoperable aortic aneurysms is appropriate. Nitro-glycerin and aconite for the relief of cardiac tension; absolute rest in bed, with potassium iodide in suitable doses to favor fibrin deposit, may properly be employed with hope if not faith. In these cases, as in others, the muscular power of the heart may be conserved, the circulation maintained, and the final failure delayed in a very marked degree by means of pneumotherapy.

MORBID GROWTHS AND PARASITES.

Morbid growths and parasites of the heart are of pathological rather than clinical interest, since they can never be recognized with any certainty, nor would they be amenable to treatment if diagnosticated. Syphilitic disease is discussed separately (page 470). Fibrosis, being essentially the reparative stage of degeneration, is included under that head. Tuberculosis is also omitted, as being always part of a general disease (see Vol. I. page 808).

Pathology.—Of the remaining new growths, (1) malignant disease is the most frequent and important, yet still exceedingly rare, and when present is, with doubtful exceptions, secondary. The primary growth may be in any part of the body, but most frequently is in the mediastinum or lungs, when the heart becomes involved by continuity. As a primary or secondary growth its relations to age are the same as for cancer in general. Its greater frequency in males may possibly have some relation to heart strain.

All forms of malignant growths—carcinoma, epithelioma, colloid cancer with melanosis, as well as sarcoma—have been found in the heart. As secondary growths they usually appear in multiple nodules beneath the peri- or endocardium rather than in the muscular substance. When the heart becomes involved by extension the muscle is more deeply implicated, and considerable portions of the cardiac wall may become extensively infiltrated with cancerous elements. Pericarditis is always present when the disease approaches the surface, and similarly endocarditis follows protrusion of the tumors into the cardiac cavity. When the new growth is situated at the base the valves may become involved, and incompetence result directly or from mechanical interference. If secondary to distant growths, the disease may affect any portion of the heart, but even then is more frequently found on the right side, as is the case when it results from extension. Usually it appears as a diffuse nodular infiltration, but in some cases it is found to have followed along the course of the veins. These cancerous foci manifest the usual tendency to undergo central necrosis, and, if life is sufficiently prolonged, they pass on to ulceration and rupture into the pericardium or into the heart cavities, with the production of multiple emboli. The colloid and melanotic forms are most frequent, and epithelioma least so.

(2) Cases of true fibromata of the heart are to be found among the records of rare pathological conditions, but such growths never present symptoms which permit of their recognition during life.

(3) Lymphomata may appear in the heart when the disease is general, and the accession of cardiac symptoms when these tumors exist elsewhere might suggest a diagnosis. They, as well as fibromata, are so rare and irregular in development as to render any special description needless.

(4) Of the other non-malignant growths, myomata are the most infrequent.

(5) In connection with obesity and the deposition of fat about the heart lipomata may develop beneath the endocardium or pericardium. When on the surface they are less distinctly separated from the general mass of fat which covers and infiltrates the heart muscle. Beneath the

endocardium they appear as distinct tumors, very rarely as large as a bean. What little they may affect the circulation cannot be important, and the danger of their becoming the starting point for thrombosis or fibrous deposits is very slight, since the endocardium is not injured.

(6) The occurrence of simple cardiac cysts is very doubtful. The origin of abscess within the heart wall as the result of acute myocarditis has been referred to in connection with cardiac aneurysm. In some of the more acute cases the abscess opens into the ventricle or pericardium, and death follows before an aneurysm can form. Their clinical history is with myocarditis. Cysts from softening without suppuration may develop from myocarditis in areas of fatty degeneration, at the seat of intramural hemorrhage, or in new growths, and remain unruptured until death comes from other causes. Such softening is most common in gummata.

(7) Relatively, and considering the relations of the organ to the usual channels of invasion, hydatid disease of the heart is quite frequent. The percentage has been put as high as 3.5 of all cases of hydatids in man. The cysts are located a little oftener in the right than in the left ventricle, and in about 10 per cent. of the cases in the septum ventriculorum. They start in the muscular tissue, but necessarily protrude into the ventricle or pericardial cavity as they attain any considerable size. This protrusion may be such as to allow the cyst to hang in the cavity as a polypoid tumor. At times they are three or four inches in diameter, but are more commonly the size of a walnut. It is seldom that the tumor produces peri- or endocarditis, as the growth is exceedingly slow and not attended by inflammatory action. Hydatids of the heart terminate by rupture or degeneration and absorption. In the former case opening into the pericardium will be followed by pericarditis, which is not necessarily, although probably, fatal. When the rupture is internal there is an inevitable embolism at some point, which may be at the valvular opening, in a main arterial trunk, or in smaller branches of the pulmonary or general circulation. Less frequently there is coincident rupture in both directions, or the internal opening is made subsequent to that into the pericardium. When double rupture happens at one time, there is multiple embolism with hæmo-pericardium and immediately fatal results. But when the cyst has discharged its contents into the pericardium before the internal opening is made, the hæmo-pericardium may develop slowly and the cyst contents become entangled in the blood clots. The final result, however, is only delayed, not changed. Degeneration and absorption are assumed from the changes which hydatid cysts are known to undergo in other organs rather than known to happen.

(8) Trichina and possibly some other entozoa may invade the heart, where they pass through the usual changes characteristic of their growth.

Symptoms.—None of the conditions above enumerated present characteristic or hardly suggestive indications of their presence. In a large proportion of the cases even, in which malignant disease had involved considerable portions of the heart wall, no symptoms were present. In any case only the most general signs of heart disease can be looked for. Among these pain is perhaps the most prominent. It may be constant,

neuralgic, extending to the shoulder and arm, or in the form of anginal seizures. As the heart becomes weakened dyspnœa and palpitation will be present. The organ is seldom so enlarged by malignant disease as to give an increased area of dulness, but the deeper pain is often associated with surface tenderness and itself increased by percussion.

The PHYSICAL SIGNS of pericarditis are usually present before the disease has become extensive. Cardiac murmurs are not constant or significant, although frequently present. When the disease is by extension and has previously been recognized in the lung or mediastinum, the above symptoms assume definite significance. The termination is frequently sudden, even in cases which have presented no symptoms and where the cause of death is known only post-mortem. The symptoms of the various non-malignant growths are even less definite. Fibromata, lymphomata, and lipomata are rarely even suspected; they can never be diagnosticated. Hydatids of the heart alone occupy a similar position. In case of their recognition in other organs a persistently increasing cardiac murmur with disturbed heart action would justify the suspicion that the heart had become involved. As in malignant disease, death is frequently sudden and comes without warning.

In a severe or even moderate case of trichinosis the cardiac symptoms will be so severe that but little increase would follow invasion of the heart muscle by the parasite. And as such invasion could cause no murmurs or surface changes, implication of the heart even when the general condition is known is rather less easily determined than in malignant disease.

PROGNOSIS.—The non-malignant growths are seldom the direct cause of death. Cancer has but one termination, and hydatids are usually nearly as fatal. In any case where a probable diagnosis has been made the prognosis must be unfavorable.

TREATMENT.—The purely symptomatic treatment of the foregoing conditions is not modified by the fact that some are as certainly fatal as others are benign. When malignant disease has reached the heart the remotest possibilities of serotherapy even cannot so much as suggest hope.

HYDRO-PERICARDIUM; PNEUMO-PERICARDIUM; SYPHILIS OF THE HEART; WOUNDS OF THE HEART; FOREIGN BODIES IN THE HEART.

By CHARLES E. QUIMBY, M.D.

HYDRO-PERICARDIUM.

Definition.—Hydro-pericardium as a condition demanding special consideration must be held to include both inflammatory and non-inflammatory accumulations of fluid in the pericardial sac. It is probable that in health there is a small amount of free fluid in the pericardium, which is augmented somewhat just at the time of and immediately following the death struggle. The amount must reach several ounces before its recognition clinically becomes possible or the mechanical effects render it pathological.

Etiology.—The non-inflammatory effusions depend upon two sets of causes, the mechanical and the systemic. The fact that fluid is not always found post-mortem in the pericardium, and that, as a rule, the amount is increased in proportion to the length of time the examination is delayed after death, seems to show that under favorable conditions even a small increase in vascular tension may cause pericardial effusion, while the infrequency of large effusions during life, even when there is considerable rise in blood pressure, similarly proves that mechanical strain is rarely the sole— and probably not the predominant—cause of those cases in which hydro-pericardium becomes pathological. It is true that the condition does occur in emphysema, pulmonary fibrosis, compression of lung from pleuritic effusion or tumors, and when there are pulmonary congestion and venous obstruction from mitral disease. But it is equally true that most of these diseases usually pass to a fatal termination without any appreciable degree of hydro-pericardium. Still further, when dropsy of the pericardium does occur with venous congestion due to disease of the heart or lungs, it is an early symptom —a rapidity of sequence that is entirely at variance with that in mechanical effusions in other locations. It is, moreover, difficult to believe that either the venous or lymphatic circulation of the heart alone can be so specially obstructed as to cause a pericardial effusion to precede general dropsy. The conclusion thus seems unavoidable that something more than high tension in the pulmonic circulation must be present before pericardial effusion will occur. That this condition may accompany atheroma of the coronary arteries seems to indicate that nutritive changes in the pericardium afford the predominant and excit-

ing cause of cardiac dropsy even when obstructed circulation is the more obvious, as is the case with mediastinal disease, tumors, and enlarged glands, or fibrous bands either within or without the pericardium.

The older theory of a *hydrops ex vacuo* only excites surprise that it should ever have been advanced.

The etiological relations of the various hæmic causes of dropsy to hydro-pericardium are more direct. The origin of the hydræmia appears to be of little import. The causative disease may be in the liver, spleen, kidneys, alimentary tract, or lymph structures. It may be simply functional, producing toxæmia; degenerative, as in hypertrophic cirrhosis; or of specific origin, as in cancer, tubercle, and syphilis. It bears the same relation to the cardiac effusion as to the general dropsy which so constantly accompanies it. Hydro-pericardium, however, is much less frequent in hydræmia than are effusions into other serous cavities, and when present is a late rather than early condition. By far the larger number of cases will be found to depend upon inflammatory changes, either acute or chronic. The former are considered under Pericarditis (page 357). In the latter the processes are of an extremely low grade, often being little more than nutritive changes in the epithelium, yet sufficient to demonstrate the local nature of the cause. Such an inflammation may be regarded as the cause of an hydro-pericardium when none other can be detected. When it occurs in connection with a general and prolonged pulmonary tuberculosis, mediastinal or pulmonary malignant disease, or in the third stage of syphilis, the specific disease involving the pericardium may be regarded as the cause.

Pathology.—The presence of even two or three ounces of straw-colored fluid in the pericardial sac when autopsy is delayed some hours cannot be considered pathological. Those collections which have formed during life are found to vary greatly in composition. While blood serum forms the basis in all cases, the other elements depend upon the cause of the effusion. The presence of fibrin in small amounts does not of necessity indicate an inflammatory origin, but when such has been the cause the amount of plastic matter will be proportionately large. Urea and other extractives are found, particularly when nephritis exists, as are biliary pigment and acids in connection with jaundice. The simpler effusions are clear or slightly opaque, yellowish in color, and but moderately glutinous. An admixture of blood, which is more frequent in cancer and tuberculosis, may render the fluid entirely opaque and of a reddish tint, deepening to a dark brown according to the proportion present. Desquamated epithelium, which is present in all chronic cases with some leucocytes, renders the fluid turbid, while the presence of larger amounts of pus cells changes the color to cheesy yellow or even green, and is associated with infection.

The outer layer of the pericardium is constantly dull and opaque, and may even appear eroded where the epithelium is lost. Both layers are pale, especially if the sac has been greatly distended. Occasionally in subacute inflammatory cases small patches of plastic exudate may adhere to the surfaces, and it may be difficult to determine whether the inflammation was precedent or subsequent to the effusion. Rarely in chronic cases with small effusions the pericardium may be thickened; but more commonly, and particularly if the sac has been firmly dis-

tended for some considerable time, the fibrous layer is thinned and the epithelia are swollen, granular, and degenerating. The membranes may also appear puffy from œdema of the subserous cellular tissue. Prolonged distention is followed by absorption of the subserous fat around the heart, with relaxation and softening of the muscle. In extreme cases the lungs are compressed and forced back and the diaphragm is depressed.

Symptoms.—In the milder cases with only moderate effusion there are no subjective symptoms, and when the fluid accumulates slowly it may attain to a considerable amount before it so disturbs the heart action as to cause the patient to complain of a growing dyspnœa, easily increased on exertion, palpitation, and præcordial oppression.

In the acute cases of albuminuria following scarlatina, and even in chronic nephritis, the effusion may be rapid, when the cardiac oppression will be severe and all the above subjective disturbances greatly intensified. As compression begins distinctly to affect the heart action it will be indicated by a lowered arterial tension, weak pulse, cyanosis, and a decreased urinary flow.

Physical Signs.—On physical examination the signs are those of pericarditis with exudation, minus the friction sounds. It is only in the extreme cases that the effusion produces any changes in the outlines of the thorax, and they are seldom appreciable, except in children or women with elastic chest walls. Such as occur start in an elevation or prominence of the thorax in the region of the apex beat, that gradually extends upward and to the left as the fluid increases. On palpation the cardiac impulse is feeble, indistinct, or entirely unappreciable. Percussion shows an increase of cardiac dulness, first noticed in the horizontal plane of the apex, and later developing upward, but constantly maintaining its greatest breadth at the base. As the pericardium begins to be distended, or certainly before the apex beat disappears entirely, the area of dulness will be found to extend beyond the point of impulse, both to the left and below. In some cases the peculiar triangular outline of dulness can be somewhat varied by changes in the position of the patient. On auscultation the heart sounds early may not be appreciably modified, save by the underlying disease, but later they become indefinite, distant, or entirely lost; but no murmurs should be present. A friction sound can exist and the case be regarded as one of primary hydro-pericardium only when the effusion has developed in connection with old pericardial thickening and adhesions. The writer has seen one such case in which the adhesions resulted from pericarditis complicating smallpox, and were known to have existed for a number of years.

Diagnosis.—The presence of pericardial effusion cannot be detected with certainty until it amounts to several ounces. But owing to its generally slow development, the physical signs are usually quite distinct when the patients first make complaint of subjective disturbances, unless repeated physical examinations have been made in the course of such diseases as are known to cause it. The subjective symptoms aid diagnosis only as they are taken in connection with the physical signs, which are essentially conclusive. The same differentiations will arise as in pericarditis with effusion, and are determined in the same manner.

When the question of diagnosis between hydro-pericardium and pericarditis arises the distinction is more pathological than clinical, and must depend upon the presence and character of a friction murmur and the constitutional signs of inflammation, since an acute development is quite as possible with hydro-pericardium as in pericarditis. In doubtful cases, when the presence of fluid would justify aspiration, the needle may properly be employed as a means of diagnosis.

Prognosis.—Even small amounts of pericardial effusion in connection with any of the causative diseases must add to the gravity of the prognosis. It is when the effusion interferes mechanically with the heart action that it specially modifies prognosis, making it exceedingly unfavorable, not because the difficulty cannot be temporarily relieved, but because it indicates an underlying condition which is not affected by the withdrawal of fluid.

Treatment.—The etiological relations in any given case of pericardial dropsy must determine the nature of all prophylactic measures. Direct treatment is demanded only when the fluid acts as a mechanical obstruction to the heart action. In some acute cases this may imply very energetic action. Theoretically, blisters or dry cups over the præcordium are indicated as soon as the effusion is detected. In some cases they may have a definite value, and more probably when inflammatory processes are also present. In chronic cases and those arising from hydræmia they are of little or no use. Temporary relief, that is also sometimes permanent, may be certainly obtained by aspiration, which should always be employed when the symptoms become urgent, care being had, however, not to attribute to compression the cardiac manifestations of general exhaustion. The operation is particularly commended when there is hope of relieving the causative disease, as in acute albuminuria of scarlet fever. When hydro-pericardium is the last addition to a general dropsy, aspiration can delay but briefly the fatal result. The operation is performed as for pericarditis with effusion (page 370).

PNEUMO-PERICARDIUM.

The presence of gas in the pericardium is so constantly preceded or followed by an inflammatory exudate that pneumo-pericardium and pneumo-hydro-pericardium may be regarded as presenting only different stages of a necessarily complex pathological process.

Etiology.—The direct entrance of gas from without through a solution of continuity in the pericardial wall forms the initiation of pneumo-pericardium in the large majority of cases, even when the consecutive decomposition may determine its continuance. The multiple possible relations of traumatism and wounds of the chest wall to pneumo-pericardium are self evident. The special forms will be considered in connection with wounds of the heart. It should be said here, however, that the entrance of air is by no means a necessary consequence of penetrating wounds of the pericardium. But both punctured and

incised wounds may involve the pericardial sac in a septic inflammation that develops a pneumo-hydro-pericardium without the advent of air. Communication between the pericardial cavity and either the air or a source of gas formation may be established by ulcerative processes. These may take place from within outward when there is suppurative disease of the pericardium, and similarly by ulceration burrowing pus may invade the pericardium. It may happen that the primary centre of suppuration contains no gas, but that the advancing ulceration extends in two directions and reaches a gas-containing cavity as well as the pericardium, as in a reported case in which an abscess of the liver opened into the stomach and pericardium. Mediastinal suppurative disease is particularly apt to form such a double connection. Pyo-pneumo-thorax, abscess of the lung, ulceration in the œsophagus, ulcer of the stomach, as well as new growths in the mediastinum, have all been noticed as the starting point of the ulceration which eventually opens the pericardium to the entrance of air or gas. The possibility, if not probability, of any advancing suppuration within the thorax or abdomen having a similar result must be recognized. The formation of gas as the result of decomposition in a septic exudate and the production of pneumo-hydro-pericardium without an opening in the sac have been generally accepted as possible, even if of doubtful occurrence, although strenuously denied by some excellent authorities. The denial is based in part on chemical analysis of gases obtained from cases in which a local generation seemed probable upon clinical examination. It must be admitted that such cases are very rare, as are those of pyo-pneumo-thorax without perforation. Nor can failure in either case to find an opening be taken as conclusive evidence that none has existed, any more than the escape of air at some point on the pulmonary surface under such pressure as is usually employed to make the test demonstrates the existence of an opening ante-mortem.

When gas is found in the pericardium without injury or evidences of destructive changes in any of the adjacent tissues, it is difficult to deny its local formation. And the fact that gaseous decomposition is known to occur in rare instances in the cellular tissues without wound of the skin renders the acceptance of this as a cause of pneumo-hydro-pericardium more easy.

PATHOLOGY.—Entrance of gas or air within the pericardium is so certainly preceded or followed by suppurative inflammation that practically a pure pneumo-pericardium never occurs. When the pericarditis is primary the character of the infection and the exudate is determined by it. In case the entrance of gas is the cause of the inflammation the appearance of the resulting exudate may be somewhat modified. More or less blood is usually mixed with the inflammatory fluids in cases of traumatic perforation, as in case of wound or puncture from a fractured rib. When an abscess opens into the sac its contents will be mingled with the inflammatory products. Broken-down liver cells, cancerous elements, pulmonary elastic fibres, hydatids, or tubercle bacilli may thus be present at different times.

Chemical examinations of the contained gases have shown carbon dioxide and sulphuretted hydrogen to be the most common components aside from the elements of the air. The former are usually pres-

ent, however, in very small percentage. The pericardial surfaces present the usual appearances of serous membranes in suppurative inflammation. The anatomical relations of the pericardium are such that a free communication is seldom maintained between its cavity and a source of air supply. Thus with a valvular opening respiratory and cardiac aspiration may gradually cause gaseous distention of the sac, or the same condition may result in even higher degree when the gas is formed by decomposition. Compression of the lung and displacement of the diaphragm occur proportionately to the pericardial distention, as in hydro-pericardium. On account of its specific gravity the gas maintains a position at the highest part of the cavity.

Symptoms.—The subjective symptoms arising from the pneumo-pericardium, *per se*, are those of general cardiac oppression, but, owing to the elasticity of the gas, they are seldom so severe as in hydro-pericardium or pericarditis. Moreover, the forces causing effusion or exudation are not antagonized by tension in the sac, as is the case in pneumo-pericardium when the gas has come from without. Indeed, so long as there is an opening in the pericardium even respiratory aspiration cannot raise the endo-pericardial pressure above that of the atmosphere. In case, however, of closure of the opening and local gaseous formation this pressure may be higher. Dyspnœa is an early symptom, and increases in proportion to the cardiac compression. Epigastric or præcordial oppression, with a feeling of indefinite anxiety, may be constant. If the onset is sudden and the amount of gas large, these symptoms will be accompanied by those of shock or collapse: the face becomes pale, the lips blue, the surface cold and perhaps damp; the pulse is weak, small, and irregular in both rhythm and force; the dyspnœa becomes intense; the anxiety is marked by great restlessness, and may be attended by recurrent periods of delirium or unconsciousness. Constitutional symptoms will vary according to the cause. Chills and fever quite certainly attend the development of the suppurative inflammation whether it precedes or follows the pneumo-pericardium.

Physical Signs.—The physical signs are easily recognized and distinctive.

On *inspection* an increasing fulness of the chest may be apparent as the pericardium becomes distended, which later becomes a marked prominence, especially in cases with soft costal cartilages and flexible ribs. This prominence is not pointed, but rather uniform over the præcordial areas and for some distance beyond.

On *palpation* the apex beat is often indistinct, but may be varied in force by changes in the position of the patient. In the horizontal position on the back it is usually entirely absent. Frequently a fremitus may be obtained from the motion of the associated exudate or effusion as it is agitated by the heart action.

On *percussion* the note varies according to the relative amounts of air and fluid and with changes in position. With the patient erect cardiac dulness is changed to flatness over the lower area, and increased laterally, particularly to the right. At the left it may extend beyond the apex beat, and its inferior limits lie below that point. Passing upward, the note rather abruptly alters from dulness to tympanitic in quality, with a pitch depending upon the size of the air cavity. With

primary pneumo-pericardium this may be different. The lower areas will then give the usual cardiac dulness, passing more gradually but sooner to tympanitic, or the entire cardiac area may give a slightly tympanitic quality of percussion.

The general outline of the combined areas will be roughly triangular, as in hydro-pericardium, but with rather a broader apex, except in pure pneumo-pericardium, when it is more nearly quadrilateral. Cracked-pot resonance has been observed, and assumed to indicate a free communication with the trachea. Changes in the pitch of the tympanitic note are said to be produced by the action of the heart. It is true that such action may alter a very little the size of the pericardial cavity, and possibly the density of its contained gas. Yet it is evident that any consequent modifications of the pitch of the percussion note must be slight, and best developed when the pericardial gas is in free communication with the air. They must depend very largely also upon the patient's position and the ratio between gas and fluid in the pericardium, for all the results of percussion are sharply modified by change in position. The area of tympanitic percussion may be decreased, or perhaps made to disappear entirely, by bending the body forward. The same area is developed to its fullest limits by the recumbent position. At times the amount of gas is so large as to underlie all the area of cardiac percussion and thus obliterate præcordial dulness.

Corresponding changes in the relations of areas giving tympanitic and dull percussion notes may be obtained in the lateral and intermediate positions, and, as a rule, are quite as easily recognized as in cases of pneumo-hydro-thorax.

Auscultatory signs are equally definite and clear; at times their intensity is remarkable. They are frequently a source of annoyance to the patient, and may be so loud as to be heard clearly by persons standing at some distance. The air space acts as a resonator for all the sounds, and not only intensifies them, but gives to each and all a peculiar metallic quality. This is due to the fact that every resonator has what may be described as a selective power by which its intensifying action on composite or compound sounds is greatest upon such portions of those sounds as correspond to its own fixed pitch. From among these the same note is therefore always made prominent, and the subsidiary harmonics remain fixed, so that all the sounds thus acquire a common quality and may even have a uniform pitch. The metallic quality indicates intensification of the even harmonics. The effect of this action on the heart sounds is peculiar, as they are less complex than others that may be heard. Not only are they loud and clear, but they may sound like a chord of several distinct notes with a peculiar bell-like quality, and this quality may remain even when the fluid exudate is sufficient to muffle their intensity. Occasionally the adventitious sounds may be so loud as to obscure the heart sounds. These new sounds are the result of agitation of the fluid by the heart action, and correspond to succussion as produced in the pleural cavity. They are so constant, however, and usually so loud and abundant, that shaking the patient, as is done to obtain succussion, seldom adds or subtracts anything appreciable. A splashing sound usually predominates, but the breaking of air-bubbles and the dropping from the upper walls add

crackling and snapping or tinkling sounds which are very characteristic. As stated above, these all acquire a peculiar metallic quality from resonance. The great variety as well as the quality and persistence of these sounds has led to their being called "water-wheel sounds," a name which was more appropriate and suggestive in the days of "overshot" wheels than in this era of turbines. Friction murmurs in these cases are rare, as moderate tension in the contained gas serves to separate the pericardial surfaces, and when present they, as well as complicating endocardial sounds, are often obscured by the intensity of the adventitious sounds.

Complications.—Pneumo-pericardium is itself usually a complication of some other disease, and primary only when of traumatic origin. As developed secondarily, it has but little influence in determining further complications. If the gas is formed *in situ*, distention of the pericardium may result in rupture, with extravasation of the gas and fluid into the adjacent parts. The relief to cardiac action is only temporary, and usually such rupture occurs just before or at the time of death. When pneumo-pericardium is of traumatic origin and primary, pericarditis is inevitable, but may be moderate and end in recovery, possibly without suppuration. But with all suppurative cases a fatal termination may be expected. Before this happens the suppuration may invade by ulceration any adjacent tissue: more commonly the opening is into either the mediastinum; œsophagus, through which the discharge is by the mouth or stomach; pulmonary tissue and bronchial tubes; or pleural cavity, with the production of a pyo-pneumo-thorax. The termination is usually by exhaustion, sepsis, or heart failure. Occasionally mechanical compression seems to be the exciting cause of arrested heart action, or in prolonged cases softening of the muscular wall is followed by dilatation and possibly rupture.

Diagnosis.—It would be difficult to overlook a case of pneumo-pericardium. Attention will certainly be directed to the heart, and the physical signs are sufficient for a diagnosis. An hypertrophied heart acting on a distended or dilated stomach may produce similar sounds, but they are plainly located below the diaphragm, as are the percussion evidences of gas, and the area of cardiac dulness is not displaced. This area of dulness also is above, not below, the tympanitic area, and their relations are not changed with the patient in a horizontal position. With distended stomach the splashing sounds are greatly diminished when the patient is erect, while the tympanitic resonance of the clear heart sounds is increased. Large pulmonary cavities near the heart, especially when the pleura and pericardium are adherent, may afford similar metallic splashing sounds that are rhythmical with the heart beat. But, as before, the area of cardiac dulness is present and of normal or increased size, even if slightly displaced. The apex beat is but little if any displaced, is full and strong, and not affected by position. The relations between tympanitic and dull areas also are constant.

The râles and metallic sounds may be modified and are intensified by cough and full inspiration. The differentiation is less easy when an encapsulated pneumo-thorax is near the heart, as adhesions are very certainly present. The pleuritic cavity will also contain fluid, and, if

the communication with the bronchial tubes has become closed or is valvular, the difficulties are increased.

In pneumo-hydro-pericardium the apex beat is within the area of changeable percussion, and its force and distinctness are modified with such changes. This is the case in encapsulated pyo-pneumo-thorax only in very rare cases. In the former the apex beat is not usually displaced; in the latter adhesions frequently draw the heart to the left. Pneumo-thorax may involve the præcordial area at its margin, but does not give tympanitic percussion over the sternum, as does pneumo-pericardium. Change of position in pneumo-thorax alters the relations between tympanitic and dull percussion, but does not change the note at and about the apex beat. When pneumo-thorax communicates with the bronchi, respiratory sounds are transmitted with metallic quality, and even if the opening is valvular or has become temporarily closed, a forced inspiration may develop characteristic sounds of its opening. This may happen in pneumo-pericardium, but is exceedingly rare. An encapsulated pneumo-thorax near the heart is always associated with pleuritic changes extending laterally. Pneumo-pericardium more frequently follows or is attended by mediastinal disease, and the associated symptoms of disturbed circulation will be much greater than in pneumo-thorax.

TREATMENT.—The treatment of traumatic and primary cases is better considered in connection with wounds of the heart. Narcotics are always indicated for the pain and general distress, and, in cases made hopeless by other conditions, should constitute the sum total of treatment. But if the cardiac condition becomes a factor in prognosis, the indications are threefold: (1) to relieve mechanical cardiac oppression; (2) to mitigate the secondary pericarditis; and (3) to sustain the heart action. The first object may be attained by aspiration of the pericardium. The form of aspiration will depend upon the peculiar conditions of each case. If air or gas still has access to the pericardium, little can be gained, except by removal of the fluid, unless the opening is valvular from without. In such cases the fluid should be removed first, and then aspiration of the gas made in such manner as to leave a permanent channel of exit. For the removal of gas the puncture is made with the patient in a recumbent position at the most prominent point in the area of tympanitic percussion. The operation is without danger if the trocar is entered slowly. The fluid is removed as directed in Pericarditis (page 370).

To allay inflammation the usual treatment for pericarditis may be employed until it becomes evident that septic decomposition has begun, when free opening and antiseptic treatment are indicated. The results of antiseptic surgery permit this to be undertaken with little anxiety, and in the assurance that it affords the patient the largest chances for recovery. As cardiac tonics those which relieve the circulation and sustain the nervous irritability are most useful. Strychnine and glonoin should be employed at first, and later, as the heart muscle is weakened, strophanthus, digitalis, and alcoholic stimulants.

SYPHILIS OF THE HEART.

THERE are few more insidious manifestations of syphilis than those which appear in the heart, not only because they are usually of the tertiary form, but because they so frequently affect the muscle without implicating the valves. Relatively, syphilis of the heart is rather frequent, considering only cases in which it is clearly recognized, and omitting many in which lesions similar anatomically cannot be traced to any clinical history of infection. It is usually associated with syphilitic changes in other organs, which, however, may be equally obscure and are often overlooked clinically.

ETIOLOGY.—The primary infection may be either inherited or acquired, and the frequency with which the heart is affected seems to bear no special relation to the manner in which the infection is received. Indeed, there are no available facts indicating the forces that determine the location of syphilis in the heart or elsewhere.

PATHOLOGY.—More commonly the muscular walls are the parts affected, but the valves, and particularly their bases, may be involved coincidently, or even, in rare instances, be the sole seat of the disease in the heart. Usually there are several foci of new growth, which may continue distinct or become merged into one mass of infiltration. The process starts in the intermuscular tissue as a cellular hyperplasia and infiltration, which results in varied anatomical appearances according to the distribution of the growth and its subsequent course of development. In one form the infiltration is diffuse and, as a rule, goes on to fibroid formation. There are thus developed firm, tough whitish patches scattered through the heart wall, or areas that are filled with fine and coarse ramifying fibrous bands. As the primary infiltration is more localized, an indurated, thickened band or hard, warty nodules may be found among the muscular fibres. The cellular hyperplasia often extends to and into the endo- and pericardium, causing thickenings and adhesions of these membranes. Total and permanent organization, however, is not the rule in these new growths. When they are well developed, or even during their growth, varying degrees of degeneration appear at different points and are indicated by spots of yellowish color. This change may go on to disintegration, and portions of the new growth, as well as the implicated muscular tissue, which has suffered similar changes, may be removed by absorption. However sharply limited the fibroid patch may appear on gross examination, it will be found to extend in fine prolongations in all directions among the muscular fibres, different patches often being connected by these bands.

In the second form the cellular growth is not only more localized and circumscribed, but more abundant, forming small nodular tumors, the true syphilitic gummata. These growths are located preferably in the thicker walls of the ventricle about the apex. The tumors are usually small, lying under the pericardium or within the wall, where they are recognized only on section. When newly formed they are rather firm and elastic, and may even seem fibrous on section. They are not vascular or succulent, and present a homogeneous slightly yellowish white surface when cut. As they grow older the outer layers tend somewhat to organization, or, if not, at least retain a longer vitality, while

the central portions undergo degeneration and necrosis, softening into a puriform or cheesy mass. When the surface elements maintain their integrity the central softened mass may again harden by desiccation, and remain as a firm cheesy or calcareous nodule, or it may undergo absorption, followed by contraction and shrinking of the nodule. Less frequently organization is complete in the outer layers by which the central mass is permanently encapsulated. These are the more favorable changes. On the other hand, and especially when the cardiac are among the earlier tertiary changes or the disease escapes recognition and treatment, the degenerative processes which are so characteristic assume the form of acute necrosis with suppuration, and extend centrifugally until an opening is made into the pericardium or heart cavities. The process thus becomes etiological to embolism, thrombosis, pericarditis, or cardiac aneurysm. Either form of the growth may occur when the valves are affected, but in this location the fibroid is more common. It results in contraction of the valvular openings, thickening and retraction of the valves, and more rarely in valvular abscess, aneurysm, or perforation. Obstruction and incompetence quite certainly follow, and are peculiarly serious conditions when the cardiac wall is also involved. The myocardial inflammation which attends all forms of the disease is usually chronic in form, tending to fibrosis or atrophic dilatation. But occasionally, and especially when the aorta is coincidently affected, an acute endocarditis and aortitis accompany a similarly acute myocarditis, in both of which there is a marked tendency to terminate in acute dilatation. Syphilitic endarteritis of the cardiac vessels frequently accompanies the lesions of the wall, or it may be the predominant, if not sole, evidence of the process in the heart. It often causes thrombosis or infarction with their usual sequences, or the more gradual obstruction of the myocardial circulation may induce more extensive and disseminated nutritive degenerations, followed by dilatation, aneurysm, or rupture.

Symptoms.—It is only when such symptoms as would naturally result from some combination of the pathological changes just enumerated are supported by a syphilitic history that the nature of the cardiac disease can be suspected, and this suspicion must be confirmed, in a measure at least, by the results of treatment before it can assume the form of a diagnosis. Some cases which have fallen under the author's observation justify the assertion that careful and frequently repeated examinations of the heart should be made during the treatment of all stages of every case of syphilis. It is especially wise to determine the existence of any heart murmurs during the primary stage, to avoid difficulty later in distinguishing new from old murmurs, despite the fact that the valves are not usually affected. Valvular lesions, however, when present, may be detected earlier in their course than mural growths, and their injurious effects consequently reduced to a minimum. Some cases of cardiac syphilis meet a sudden death without having suffered sufficiently from symptoms referable to the heart to have directed attention to that organ. Very often, however, cardiac symptoms are prominent, and these cases will appear in two classes, dependent upon the location and character of the new growth. When-

ever the disease is intramural the symptoms will quite certainly develop slowly, becoming prominent only when the degenerative changes are more or less advanced, and will be subjectively those of lessened cardiac force, with physical signs of dilatation either uniform or aneurysmal. To these are added later the symptoms of pericarditis as the growth approaches the surface. Unless the valves are involved the progress of the disease is marked only by changes in the intensity of these symptoms and the gradual appearance of the results of impeded venous and weakened arterial circulation. Most frequently this class of cases terminate suddenly. Very rarely these same intramural growths give rise to acute myo- and pericarditis, of which the symptoms are the same as when dependent upon other causes. In the second class of cases the valves with the immediately adjacent myocardium are involved. As before, the invasion and progress are, as a rule, insidious and slow, so that attention is called to the heart only when the valvular lesions have been reflected upon the heart muscle in the form of hypertrophy or dilatation, or are made manifest, if mitral, by impeded pulmonary circulation. Under these circumstances the physical signs of valvular injury will be prominent. As they usually come under observation some years after the initial lesion, such an origin should always be kept in mind as a possibility in all cases where valvular disease is of doubtful etiology. It is in this form of the disease that acute inflammatory changes are relatively most frequent, although still absolutely uncommon. They appear as acute endocarditis or endomyocarditis. When the specific lesion is in the aortic valves, the valvular endocarditis is often associated with acute aortitis. All of these conditions present the usual symptoms.

Complications and Sequelæ.—While, pathologically, syphilis of the heart is a distinct disease, clinically it is more properly regarded as simply a cause, and the resultant conditions as separate diseases rather than as complications or sequelæ. Possibly fibroid degeneration, aneurysm and dilatation with valvular lesions may be regarded as sequelæ, since they may be persistent when all specific activity has been arrested and all new growth organized or absorbed.

Diagnosis.—The dependence of cardiac symptoms and their causative lesions upon syphilis can be assumed only from the history and the presence of similar disease in other organs. To become a definite diagnosis such assumption must be confirmed by the results of treatment.

Prognosis.—When the possibility of cardiac implication is kept in mind in every case of syphilis, practical assurance can be given that it will be prevented, as it is confined almost entirely to cases which have neglected treatment. When once developed the prognosis must depend upon the location of the lesion and the extent of the injury already accomplished. The progress of the specific process can usually be arrested, and such new growth as has not become fibrous may be absorbed. Fibrous patches are always liable to develop into aneurysms, however complete the restoration of surrounding muscular tissue. Under the processes of absorption valvular lesions are found to show signs of improvement, but seldom disappear entirely. Muscular compensation is usually not as prompt or complete or persistent as in similar

valvular changes from causes that do not affect the myocardium. With the mechanical measures now available for diminishing the injurious effects of regurgitant valvular lesions the prognosis in such cases is favorable for prolonged relief previous to extensive degenerative changes in the heart walls.

Death often follows a prolonged period of syphilitic marasmus in which cardiac disturbance very possibly has not been prominent. It is probable that here the cardiac lesion has been of rather recent development.

In many cases death has been due to rupture of the heart; in others to acute dilatation and paralysis both with and without valvular lesions, or to the results of other specific growths. While sudden death from rupture is thus not rare, it need form little part in prognosis, since it occurs almost solely in cases that have not been recognized as syphilitic, and usually have not even come under treatment.

TREATMENT.—For the arrest of syphilitic new growth and the promotion of absorption the classical treatment of late syphilis should be instituted upon the first suspicion of the origin of cardiac symptoms, and pushed to the extent of individual tolerance. Most of the cases which are definitely recognized will occur at a considerable period after the initial lesion, and such late manifestation may be assumed to depend either upon some vice of constitution, inherited or acquired, or upon defective treatment in the earlier stages. Careful attention to the general condition should therefore accompany specific treatment. In this latter the use of potassium iodide will predominate until there is reason to believe that absorption of the new growth has reached its limit, although a mild mercurial is of service at the outset. Following this, a mild course of mixed treatment should be continued persistently for a year, when, if no symptoms of recurrent trouble have appeared, the iodide may be omitted and the mercurial continued alone for a similar period. In most cases further trouble need not be expected. At the same time, these cases should remain under constant supervision, and should undergo careful examination at stated intervals of not longer than six months at first, and then a year.

In view of the uncertainty as to the extent of the disease and the dangers of cardiac rupture, all cases should be put at rest and forbidden all muscular strain until the cardiac strength is determined; and indeed special care should be exercised not to strain the heart until a good degree of muscular regeneration is assured. To assist in promoting this muscular repair a full nitrogenized diet and some preparation of iron are most useful. When the iron produces headache, its use may often be made possible and profitable by inhalations of ozone or oxygen if the heart condition forbids outdoor exercise.

To relieve the heart strain from valvular incompetency the usual cardiac tonics are of service, of which glonoin, strophanthus, and digitalis are the most satisfactory, either separately or in combination.

For several years the writer has obtained most satisfactory results from pneumo-therapy in equalizing the circulation and establishing cardiac compensation. In several cases it has entirely superseded the use of drugs. Accompanying inflammatory processes in the heart walls or serous membranes are treated as under other conditions.

WOUNDS OF THE HEART AND FOREIGN BODIES IN THE HEART.

THE developments of antiseptic surgery, with all the revealed possibilities, have conferred a new significance upon wounds of the heart and converted theoretical into clinical interest. The condition remains as a medical subject only by courtesy. Early in the sixteenth century it was recognized that wounds of the heart were not necessarily fatal, and near the middle of the century the healing of such a wound was reported. A full consideration of the subject would necessitate the report of individual cases as they appear in most widely diverse forms, but for brevity they will be classed under two heads: I. Rupture and traumatisms of the heart from external wounds, including foreign bodies which find entrance through the wound; II. Foreign bodies which reach the heart from within the body.

ETIOLOGY.—Among the instruments by which the heart has been wounded, and which include the widest variety of articles and the most unique and improbable things, knives, bullets, and needle-shaped articles are quite naturally the most common; and this statement is true in general whether the statistics are taken from civilized communities or among savage and semi-barbarous peoples. As a result, there is a broad similarity in the character of the wounds themselves. The larger number are intentional wounds, the result of attempts at murder or suicide. Among the accidental cases penetrating instruments are less frequent, and the heart lesion may be unattended by an open wound. In this class, which includes rupture from strain, some sudden or severe compression of the thorax is usually the cause. Accidents of this nature happen from premature explosions, falling masses of stone or iron, and most frequently of all from being run over by heavy vehicles. Of the same nature are elevator accidents, in which the chest is crushed, blows from the fist, kicks of animals, crushing in machinery, and blows of flying pieces from explosions of boilers and rapidly revolving wheels. In one case a stream of water was the reported cause.

Wounds of the heart, oddly enough, are found to be more frequent proportionally to other wounds in civil than in military life, even during a time of war. Among over 10,000 penetrating wounds of the thorax taken from the records of the Civil War only 4 were of the heart. Foreign bodies are often left in the heart as the result of a penetrating wound. Most commonly they consist of some portion of the wounding instrument or bits of the clothing that have been carried in. This is more apt to happen when the instrument is blunt, as a fragment from a bursting gun, rock-blasting, or something upon which the patient has fallen. In many of these cases the wound of the heart is indirect through the penetration of a fractured rib or the sternum.

In the second class the penetration is from within the body, usually by some foreign body that has been swallowed and caught in the œsophagus or stomach. Needles and pins head the list in frequency, followed by fish-bones and a few other needle-like articles.

PATHOLOGY.—The forms of cardiac injury are even more varied than their causes, and can be given only in general. The anatomical relations of the ventricles to the thoracic wall seem sufficient to account

for the slightly greater relative frequency of wounds of the right than the left ventricle. If we exclude injuries of the pericardium which do not involve the heart itself, wounds of the ventricles, either singly or together, form nearly 75 per cent. of all cardiac injuries. The relative difference between the auricles, however, is much greater than between the ventricles, the right auricle suffering nearly three times as often as the left. It is not remarkable that the pericardium should be wounded without injury of the heart, as happens in about 10 per cent. of cases.

In rare instances the heart itself suffers a lacerating or penetrating wound without rupture of the pericardium. Rupture from strain generally, and from compression or blows without penetration, frequently occurs without injury of the pericardium. In character wounds of the heart vary all the way from general laceration and complete separation from the vessels to a fine puncture which closes upon withdrawal of the foreign body without even allowing any escape of blood. In fine punctured wounds it may be difficult to detect the external opening, and upon careful dissection the track of the wound may be found to present a broken course, due a different arrangement of the muscular layers from that which existed at the time of injury. The direction of the smaller wounds has much to do with determining their importance. When oblique to the heart wall the valvular action of the muscular layers is greatest, and the hemorrhage is reduced to a minimum. Some small blood clots will usually be found at the point of penetration and just under the endocardium in all but the most minute wounds. The direction of the cut, if incised, also influences to a degree the amount of gaping. With the incision parallel to the muscular fibres contraction may nearly close a wound in that layer, which in a deeper layer gapes very widely, as it runs across the muscular fibres. In this way an internal gaping wound may be filled with clots, while the blood has not reached the pericardium.

Gunshot wounds produce the same destruction of tissue here as elsewhere. Very small bullets may possibly pass through the heart without leaving an open canal, but generally the wound is large and ragged and the extravasation of blood fills the pericardium. When the bullet is of large size considerable portions of the heart may be pounded into a soft pulp. Ruptures and contusions also give ragged wounds, which often extend the full length of the ventricle. In contusions and crushing accidents, more particularly, the entire heart may give evidence of injury without laceration of the muscular fibres at any particular point. In these cases also the pericardium will usually be filled with clots. Foreign bodies may be found at any point in the track of the wound, frequently being lodged in the pericardial sac, heart muscle, or cavities. At first they are surrounded simply by blood clots, or, if small and sharp, may be fixed in the heart muscle without hemorrhage; but when of long standing they will be found encapsulated either with or without pus. Not rarely they have been thrown off by suppuration when cases were treated upon the expectant plan. When needles or other similar articles invade the heart from the œsophagus or after prolonged wanderings in the body, they seldom become encapsulated or remain free in the pericardium, but rather tend to enter the muscle or even protrude into the cavities unless their course is such as permits them to pass

through a portion of the heart and continue their excursions. They show but little tendency to excite acute inflammation, but may remain in the muscular tissue and become surrounded by only a small amount of fibrous new growth. Repair of cardiac wounds is possible, and is by cicatrization. The nature of the wound has much to do in determining this result. The fact that a rupture of the heart is not known to have undergone repair is probably due rather to its constant occurrence either in a previously degenerated heart, or as the result of most violent traumatism that produces total rupture or other equally serious injuries, than to its being a lacerated wound. Cardiac cicatrices that are the result of acute reparative processes show more resistance to strain than do chronic fibroid growths in the heart, and they undergo progressive contraction as do other cicatrices, the strength of the wall thus being maintained as they become reduced in size. With such cicatrices the original wound is sharply outlined, but reduced in size. At other times the fibrous growth extends irregularly into the heart muscle, and results in a more or less extensive fibroid induration surrounding the seat of injury. These plates are less elastic, and more commonly become the seat of aneurysm than do the clearly defined scars. Adhesions may form between the cardiac and pericardial cicatrices.

In a few cases where cicatrization does not occur death is delayed for a considerable time by a plugging of the wound with clots. Subsequent suppurative inflammation, however, soon causes their disintegration, with reopening of the wound and fatal secondary hemorrhage. This happens more frequently when foreign bodies have been left in the wound. Very rarely in such cases the wound heals by granulation, the foreign body having been thrown off by suppuration or encapsulated with or without sinus. It should be said that the above pathology applies to cardiac wounds under former, and still quite generally prevailing, methods of treatment. The results of antiseptic surgery should in the near future necessitate many additions and render the pathology of unassisted wounds obsolete.

Symptoms.—In the large majority of the more severe, and in many of the apparently minor, wounds of the heart death is immediate or delayed only a few moments. It is attended by sudden pallor of the whole surface and complete loss of consciousness. If the heart's action continues long enough to allow an examination, the surface of the body will be found cold, the pulse small, weak, and irregular, the respirations shallow or gasping, the pupils dilated; in a word, all the evidences of extreme shock are present. Rarely excessive mental excitement may prevent or delay this shock. Cases are on record in which persons after receiving severe penetrating wounds of the heart have given evidences of shock, but have executed voluntary violent muscular motions in full consciousness for several minutes, and fallen dead while still in action. When the primary shock is less severe and death longer delayed, a fainting fit may take the place of coma, from which the patient rallies to a semi- or complete consciousness. In these cases are seen other symptoms of severe central nervous depression. Vomiting is a frequent and early symptom, and may be attended by involuntary passages. When conscious the patient will complain of a sense of great uneasiness and anxiety, and, if hemorrhage has been severe, of extreme weakness.

There is always restlessness, that often extends to trembling, with possibly chill and convulsions. Dyspnœa is constant, and a feeling of suffocation, which is especially pronounced when there has been much loss of blood or the pericardium is distended with blood, Pain is not a constant symptom, and is apt to be more severe when the wound is slight: with severe contusions or extensive lacerations sensation is obtunded.

Of the clearly objective symptoms hemorrhage is the most important, aside from the external wound, although its amount, both external and internal, is no necessary indication of the gravity of the heart wound. Very abundant external hemorrhage may come from a severed artery in the thoracic wall, and but little may appear when the pericardium is completely distended or when the blood has found entrance to the pleural cavity. Little reliance also is to be placed upon the color of the blood as indicating the seat of the wound, since, except where the flow is excessive, a goodly portion comes from the thoracic wound. In case the lungs are implicated the blood may be frothy from having air forced into it. Bloody sputum alone does not necessarily indicate that the two wounds are still in communication. Hemorrhage is least in punctured wounds and when the cause of the injury remains fixed in the wound. When scraps of metal or other small articles are driven into the heart, the external wound may appear insignificant and hemorrhage be practically *nil*. These cases also often present very different subjective symptoms. Shock is often slight or but transient. Reaction is strong and death is delayed several weeks, and results then from the reactionary inflammation—*i. e.* from peri- and myocarditis, both of which may be suppurative. Recovery is possible in all except the most severe cases, and will always be reached through inflammatory changes which afford the usual symptoms of more or less severe peri- and myocardial inflammation.

It is evident that after the first shock has passed the heart action as manifest in the pulse will present no regularity. The physical signs are equally uncertain and variable. Percussion will afford information as to the degree of hæmo-pericardium, and assist in determining involvement of the pleura. At first auscultation is unreliable. In case of penetrating wounds, if an endocardial murmur can be definitely recognized, it would be presumptive evidence of injury to the valves or their attachments, more especially if the murmur is diastolic. Perforation of the septum, and possibly protruding or adherent clots, are also productive of ventricular murmurs. Later, as recovery takes place, both peri- and endocardial murmurs are developed.

When foreign bodies enter the heart from within, symptoms are often entirely absent, and usually are few and not characteristic. Needles apparently traverse the heart, as they do other tissues, with impunity. It is presumable that in the heart they might cause some irritability of action, and that within the cavities a murmur might result. But it is only when such an article is known to have been swallowed, and its previous course has been followed, that cardiac symptoms become even suggestive.

Complications and Sequelæ.—Adjacent organs often suffer laceration or perforation when the heart is wounded, the lungs and medias-

tinum being injured most frequently in connection with stab wounds. When the cause is a blow or crushing force, the ribs or sternum are usually fractured before the heart receives serious injury. Indeed, the ragged ends of these bones frequently produce the heart wound. Pericarditis is certainly developed when death is delayed, and pneumopericardium is common. Injury of the diaphragm and abdominal organs is frequently associated with wounds of the heart, but rarely bears any dependent relation to the cardiac lesion, save in the rare cases where pericardial suppuration makes an opening downward. Embolism and thrombosis are often direct results of the injury. In cases which go on to recovery there may remain a permanent valvular incompetency or obstruction. Within the heart wall relaxation of the cicatrix may result in aneurysm, and finally rupture, or with the firmer cicatrix pericardial adhesions may seriously impede the heart action and force the patient to a life of physical inactivity. Persistent suppuration and the formation of a sinus occasionally attend a prolongation of life that finally terminates by exhaustion and myocardial degeneration.

It is somewhat surprising to find that immediate death is much less frequent than the prolongation of life for a longer or shorter time. Even in cardiac rupture, which stands first as a cause of immediate death, the fatal termination is delayed in over 60 per cent. of cases. This percentage is slightly greater in gunshot and incised wounds, while with punctured wounds not over 10 per cent. are immediately fatal. Wounds of the right heart also less often cause immediate death than do those of the left heart. This may be due in part to the lower vascular tension of the lesser circulation, which favors clotting and plugging of the wound, but must have some dependence upon the anatomical relations, which rather necessitate a more extensive wound and greater force when the left heart is reached. The duration of life in eventually fatal cases varies within wide limits. When it is but a few hours, death results, as in the immediate cases, from shock and hemorrhage to the extent of causing cerebral paralysis, or, when the pericardium is intact or the external wound closed, from compression of the heart. At a later period, extending to ten days or two weeks, recurrent hemorrhages and secondary inflammation are usually the determining causes of death. If the patient survives for several months, the end may be due to complications, particularly septic inflammations; to myocarditis and cardiac degeneration; to the rupture of a cicatricial aneurysm; or to the mechanical results of valvular lesions. Wounds of the heart undergo cicatrization in about 12 per cent. of the cases, of which nearly all are punctured or non-penetrating in character. Ruptures and wounds of the auricles are most certainly fatal.

Diagnosis.—Even in the most severe wounds of the thorax it is not always easy to decide whether the heart is implicated or not. All the symptoms of severe shock may be present, or even extensive hæmopericardium occur, without injury of the heart itself.

External hemorrhage has no special significance unless extreme. The probability of cardiac injury may be partially determined from a knowledge of the instrument causing the wound and of the entire circumstances attending its production. As a rule, exploration of the wound is not to be employed as a means of diagnosis solely, as the

danger of displacing a clot is not counterbalanced by the value of any knowledge to be obtained, yet it may be of some assistance when required for the removal of foreign bodies. Moreover, an exact diagnosis is required only as a basis for radical surgical treatment.

The profuseness, but not the persistence, of the hemorrhage is suggestive of perforation of a heart cavity.

PROGNOSIS.—Sufficient has been said in outlining the clinical course and terminations of cardiac injuries to indicate that the prognosis must always be one of possibilities when life has been prolonged sufficiently to require it. It must always be remembered that not only have extremely serious wounds of the heart gone on to recovery, but that others apparently trivial often prove speedily fatal. Primarily, the gravity of the prognosis varies directly with the initial shock and with hemorrhage, more particularly as manifested in hæmo-pericardium, rather than by external flow. Later a moderate degree of pericarditis is favorable than otherwise, but not so endocarditis.

Foreign bodies, if aseptic and susceptible of encapsulation, do not materially alter prognosis.

TREATMENT.—The first question which will demand decision relates to active surgical interference. Until recently non-interference has been the strict rule, and it is still impossible to formulate just the degree of positive surgical measures that would meet with general approval. The answer properly belongs to surgery, but it may be said here that clinical results already justify surgical relief of severe cardiac compression, removal of foreign bodies and septic matter, at least in non-penetrating wounds, excision of offending portions of fractured ribs, with suture or drainage of the pericardium as may be indicated. Suture of experimental wounds has been successfully accomplished in animals.

Medically, the indications are to arrest hemorrhage, relieve shock, and support the heart's action. At first the cardiac depression materially assists the formation of hæmostatic clots, and at this time stimulants should be withheld as far as possible, and the relief of shock deferred until clots in the wound have become fixed and firm. Shock itself is treated upon general principles as from other causes, but with a view to the possible influence of reaction in starting secondary hemorrhage. Pain and restlessness are best relieved by opium and cerebral depressants rather than by cardiac stimulants. Digitalis is to be avoided, and vascular tension lowered rather than raised. Secondary inflammations are moderated by cold applications over the heart and cardiac sedatives. When cicatrization has been obtained, all cardiac strain should be avoided for a long time. When a wound of the heart is suspected, it is wiser to assume its existence and compel the patient to maintain absolute rest until all danger of rupture or extension of the wound is past.

NEUROSES OF THE HEART.

By JAMES T. WHITTAKER, M. D.

General Considerations.—A neurosis implies a disturbance in action independent of discoverable or demonstrable organic lesion. The condition is expressed in the term functional disturbance, and has reference to derangement in molecular action, the nature of which at the present time lies largely in the domain of speculation. Without any attempt to define or describe molecular changes, perhaps the easiest conception of a neurosis is that of temporary disturbance. In a neurosis there is no permanent destruction, and what alteration is produced is capable of entire *restitutio ad integrum*. At the present time the tendency is to underestimate the influence of the nervous system in pathology, and to put in the foreground the action of the parenchymatous structure itself under the influence of toxins. Nowhere is this disposition more evident than in the case of the heart. For the heart beats of itself, independent of any connection with the nervous system, and the excised heart still continues to beat with natural rhythm. Even exsected fragments will pulsate regularly for a time. The fact is, the heart muscle pulsates as soon as it is formed and before it is supplied with nerves or ganglia. The heart in the embryo of the chicken may be seen to pulsate at a period before the nervous system is developed.

To keep up a continuous supply of fresh blood it is necessary, not only that the heart shall beat, but that it shall beat with rhythm. It might therefore be believed that the rhythmic action of the heart was secured through the intervention of the nervous system, but this is also not the case, for, as already stated, the exsected heart not only beats, but beats with rhythm. Harvey saw that the auricles contract first, and that on receipt of blood from the auricles the ventricles contract in turn and discharge the blood into the arteries. It is now known that the rhythmic action of the heart is secured by the contraction of muscular fibres which pass directly from the auricles to the ventricles. Such transition fibres have been directly demonstrated in the heart by Romberg and His.

These anatomists showed also that the ganglia of the heart, which exist only in the auricles, are connected with the sympathetic system, and that they are concerned only with sensation and have nothing to do with motion. This demonstration came something in the way of a surprise. The rhythmic action of the heart had been heretofore attributed to the stimulus of the blood itself or was ascribed to the direct influence of the nervous system.

Some of the best anatomists and physiologists were unwilling to subscribe to the new view; Pickering, for instance, opposed it. Picker-

ing studied the embryo heart under the most natural conditions—*e. g.* through an opening in the egg-shell covered by glass. Various poisons dissolved in physiological salt solution and heated to the temperature of the embryo were introduced into the heart by means of a subcutaneous syringe. It was seen in this way that muscarin had no effect upon the embryo heart, and atropine reduced its activity but very little; that the action of digitalis and of strophanthine was the same upon the embryo heart as upon the adult heart—that ether increases the activity in the heart of the embryo just the same, while chloroform paralyzes it.

Langendorff also objects to the view of Romberg and His which fixes the motion in the heart muscle alone and relegates the ganglia to reflex influences. Langendorff shows that certain phenomena which take place in the heart during suffocation and in apparent death by heating cannot be explained without appeal to a nervous centre, and calls up the fact established by him years ago that the heart muscle free of ganglia may only be made to beat under such conditions as show the persistence of artificial irritation.

Kaiser goes so far as to deny the rhythmic action of the heart muscle. The pulse as observed in the heart free of ganglia depends not upon any peculiar rhythmical motion, but upon outside influences, as in the variations of intensity in the conduction of the constant current. According to this author, the rhythmic action of the heart depends upon the regular interruption of excito-motor influences which continuously irradiate from the ganglia situated in the sinus. Engelmann refutes this view absolutely on the basis of numerous experiments upon suspended frog hearts. Accurate measurements of the conductivity of the wave show that the irritation which causes contraction passes from the auricle to the ventricle only through muscle fibres, and not through nerves. "Whoever maintains conduction by nerves must prove the existence in the frog of nerve fibres which under the same conditions would conduct irritation a hundred times slower than that in any nerves hitherto known." Important is the fact also that the muscle fibres of the auricle, even after the complete cessation of their contractility, may still conduct motor impulses to the ventricle, and with the same rapidity as if they had been capable of contraction.

Kent confirms the view of Krehl and Romberg regarding the connection of the auricle and ventricle by muscular tissue also in warm-blooded animals. Thus Kent finds in the newborn rat such a connection in the form of peculiar branched muscle cells, and was later able to establish a muscular connection in adult animals of higher class, in which the demonstration is more difficult on account of the considerable development of connective tissue in the auriculo-ventricular furrow. For instance, such connecting fibres can be demonstrated in the ape in only a few places; nevertheless, in the midst of the connective tissue there are to be found spindle-shaped or branching granular cells, which stand in connection, on the one hand, with the musculature of the ventricle, and, on the other, with that of the auricle; and, notwithstanding their peculiar form, they are known by the presence occasionally of striation to be muscle cells. This muscular connection is found especially in the higher adult mammals, while in the lower forms there is a more direct radiation of muscle fibres. It is seen, therefore, that the

contraction of the heart in warm-blooded animals is a continuous muscle wave, and it was not difficult to excite by faradic irritation in the newborn animal (whose heart remains for a long time excitable) contractions from the apex to the auricle. This contraction could be induced both in the newborn and adult animal after the heart had been brought to a standstill by irritation of the vagus. There was no difference in time between the transmission from the auricle to the ventricle and *vice versâ*. But in the newborn the time of transmission was found to be, in correspondence to the histological condition, shorter than in the adult, which speaks for the fact that the pause between the contraction of the auricle and ventricle is caused by slow conduction through scanty muscular fibre (Roether).

It may be considered as demonstrated, therefore, that the action of the heart is automatic in the muscle, and that the rhythmic action is secured by the direct transmission of an undulatory wave from the auricles to the ventricles. It was, in fact, recognized long ago that other muscular tissue is endowed with power of rhythmical contraction, for such motion has been seen in the veins and demonstrated in the hearts of lower animals.

But to supply the varying wants of the body it is necessary not only that the heart shall beat, and beat with rhythm, but that it shall also discharge at times less or larger quantities of blood. Thus the heart beats in response to the demands of the tissues more quietly in sleep, more forcibly under exercise. Provision is made for the supply of different organs in the dilatation and contraction of the arteries, which render unnecessary any appeal to the heart, but any excessive demand on the part of the tissues may be met only by increased action of the heart itself; and this regulation of the force and frequency of the heart's action stands directly under the control of the nervous system. Derangement of this control results in irregular action which becomes purposeless and finally wears out the heart itself.

The nerves which regulate the action of the heart consist of branches from the vagus, which are in turn largely derived from the spinal accessory nerve; branches also from the superior and inferior laryngeal nerves; branches from the cervical ganglia and the first dorsal ganglion; branches from the pulmonary plexus; and occasionally a branch from the descending part of the hypoglossus (Luschka). Finally, the heart has imbedded in its substance ganglia of its own, which are to be regarded, however, as stated, as component parts of the sensory sphere of the sympathetic system. Provision is thus made in the branches from both the cerebro-spinal and sympathetic system to secure motor, vaso-motor, and sensory impressions.

The disposition of the nerves of the heart is such as to secure inhibitory and acceleratory action. The vagus is the inhibitory nerve of the heart. It has to do with the regulation of the force and frequency of the action of the heart. Irritation of the trunk of the vagus disturbs the rhythm or retards the action of the heart. More extensive irritation arrests it completely, at least for a time. The sympathetic nerve fibres are chiefly concerned with sensation, but the trunk contains also motor filaments which act as accelerator nerves. Stimulation of the nerve fibres which issue from the sympathetic ganglia increases the frequency

in the action of the heart. Such stimulation is often effected by the irritation of disease processes in the spinal cord. Thus the heart beats with increased frequency in the course of tabes dorsalis, disseminated sclerosis, and progressive paralysis. When the centre of these nerves is destroyed the acceleratory influence is lost, and the action of the heart may be retarded twenty to forty beats per minute.

The ganglia of the heart which are found exclusively in the auricles belong evidently to the receiving chambers and have to do with the perception of sensation. Connected with the sphere of sensation are fibres which run in the opposite direction from the heart to the brain or cord through the trunk of the vagus. Thus the tissues may make known their wants to the heart through efferent nerves, while the heart may signify its capacity to the tissues through afferent nerves, which issue from the heart and find their way to the nerve centres. Irritation of the cardiac extremity of the superior laryngeal nerve reduces the pressure in the arteries; hence this nerve is known as the depressor nerve.

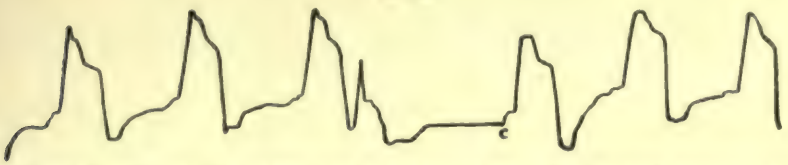
The centres for these various nerves lie, with the exception of the spinal branches of the accessory nerve, quite near together in the medulla, and are found in close association with the vaso-motor and respiratory centres. Intercommunication is easy and quick, so that the action of the heart may be regulated to meet the demands of oxygenation, nutrition, and metabolism. Any disturbance of the harmonious action which exists between the inhibitory and accelerator nerves or in the regulation of the adjustment of the heart to the varying wants of the body is expressed in irregular action, weakness, palpitation, and pain.

Arrhythmia.

The heart is a force pump which supplies the tissue with blood, but as the supply must be continuous and the heart cavities contain but a few ounces, it is plain that the heart must fill and empty itself continuously, and the discharge must be *saltatim*. So the heart fills and empties itself continuously. The various chambers open and close systematically, and the regularity of this procedure constitutes what is known as the rhythm of the heart. As stated above (page 481) the rhythmical motion begins in the auricles, and is continued by direct muscular fibres to the ventricles, the contraction of which discharges the blood into the arteries. The auricles contract simultaneously; the undulatory movement is continued to the ventricles, with an interval which is only apparent and not real, and the contraction of the ventricles is followed by a period of rest. The ventricles expand or dilate during the period of rest. The diastole occurs in rest. Under the ordinary frequency of action the contraction of the auricles takes place in 0.177 second, of the ventricles in 0.34 second, while the rest occupies 0.4 second. These intervals are all shortened under increased frequency of pulsation, but the main amount of shortening occurs at the expense of the diastole, so that rapid pulsation is effected by a disproportionate loss of the period of rest. Under slower pulsation the period of rest is correspondingly prolonged. Moreover, the interval between the contraction of the auricles and ventricles becomes real; sometimes, indeed, the auricles contract two or three times before the ventricles begin their

work. Sometimes the auricle empties itself more slowly. In either event a frustrate contraction may ensue, as illustrated in the following diagram:

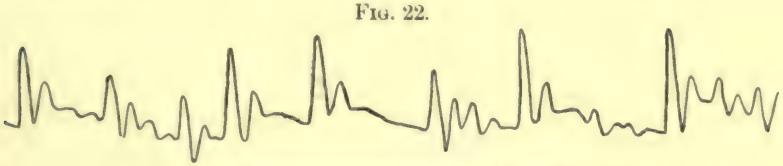
FIG. 21.



Frustrate contraction: the slow emptying of the auricle is represented in the horizontal line which terminates at *C* (Damsch).

The regularity in the action of the heart, even under the most disturbing conditions, is so distinct as to have excited admiration at all times. Nevertheless, the rhythm is not absolute. Close inspection will reveal irregularities even in the healthiest persons, though they may be detected at times only by means of some intensifying or magnifying apparatus. But these irregularities are, for the most part, trivial. As a rule, the heart beats rhythmically, even during the existence of fevers which may greatly increase the frequency of its action; and it is only in the presence of some serious disturbance of nutrition, as by mechanical interference with the blood supply to the heart from atheroma of the aorta, sclerosis of the coronary arteries, or when the heart muscle itself is poisoned by the action of some toxin, as in the course of the graver infections, that the rhythm of the heart is really disturbed.

FIG. 22.



Pulse tracing showing arrhythmia (Musser).

The simplest form of arrhythmia is represented in the loss of a beat, in the so-called intermittence in the action of the heart. But the loss of a beat of the pulse as appreciated at the radial artery does not necessarily indicate the loss of a beat of the heart. It happens that occasional heart beats are not powerful enough to propel the blood to the radial artery. Laennec long ago noticed these incomplete contractions, and Hochhaus and Quincke made recent studies of what they call "frustrate" contractions. False intermissions are easily recognized by direct auscultation of the heart. But the heart may itself lose or skip a beat, and this loss may occur every second or third beat, or at any time later, at distinct or irregular intervals. Any regular disturbance of rhythm constitutes what is known as *allorrhythmia* (another rhythm).

The arrhythmia may be a matter of time or force. Thus, the rhythm may be disturbed by the closer approximation or further separation of individual beats.

The approximation of two beats, followed by a long pause, constitutes the *pulsus bigeminus*—of three beats, the *pulsus trigeminus*, etc. A regular succession of strong and weak beats constitutes the *pulsus alternans*. The combination of a strong and weak beat, followed by

a pause of unusual length, constitutes the *pulsus bigeminus alternans*. A partial contraction, what is sometimes called a frustrate contraction, is represented in the hemisystole. When the arrhythmia is complete, so that the heart falls into a state of absolute disorder, there is said to be *delirium cordis;* or if the pulsations of less tumultuous character are so rapid as to appear as mere vibrations, the condition is known as *tremor cordis*.

The pulse should become stronger with each act of inspiration. When it becomes feebler instead of stronger, there is said to be a *pulsus paradoxus*. The contraction of the heart is here too feeble to propel the blood through the vessels in the chest, which are dilated by the act of inspiration.

Etiology.—While it is true that the rhythmic action of the heart is automatic and is inherent in the muscle tissue itself, it is also true that the rhythm may be disturbed, besides by affection of the muscles, under the influence of the nervous system. The force and frequency of the action of the heart are regulated by filaments in the vagus and sympathetic nerves; hence the regular action of the heart may be disturbed by impressions or lesions in the direct course of these nerves or by irritation reflected from distant parts of the body.

Romberg and Bruhns made a series of studies as to the cause of the disturbance of circulation in the acute infections, heretofore clinically designated as heart weakness or heart failure. Romberg had shown that in these disturbances it is not only the heart, but also the vasomotors, that are affected. It is not only the activity of the heart, but also the condition of the muscles and nerves of the vessels, as well as their centres, which are necessary to the maintenance of a normal circulation. In diphtheria, for instance, there is observed a remarkable but peculiar influence upon the heart. The capacity of the heart remains, as far as experiments show, normal. We may say, with all certainty, that the heart takes no part in the disturbance of circulation which causes death. But the heart rhythm is changed. The heart stroke is markedly retarded. The frequency sinks to 170, 150, sometimes to 132, to 120. This retardation in the action of the heart has been also seen in guinea-pigs and frogs after intra-peritoneal injection of the poison of diphtheria.

How is this disturbance in the heart rhythm to be explained? We may not find it in a lessened blood pressure. The cause lies in the heart itself. The fact that only the rhythm, not the power, of the heart is influenced by diphtheria excites the suspicion that the poison acts chiefly upon the section regulating the rhythm—to wit, the auricles, injuring the ventricles but little. Support for this view is found in the fact that the musculature of the auricle shows parenchymatous change in high degree, splitting of muscle substance, vacuolar degeneration—changes which are found in the ventricle in only slight degree. The rabbit's heart shows marked fatty degeneration, as does also the human heart in diphtheria. But this change may not be held responsible for the retardation, for the retardation may be pronounced, while fatty degeneration is shown only in isolated drops of fat in the muscle fibres. Romberg's observations show that fatty degeneration occurs only a short time before death, and his experiments support those of Krehl,

made upon the basis of an exact chemical study, that fatty degeneration and the capacity of the heart are independent of each other in large degree. It is true that the poison of diphtheria hurts the heart by the development of a diphtheritic myocarditis, but the chief damage to the circulation does not occur as the result of this lesion, but in consequence of injury to the vaso-motor system.

Studies in the infections lead to the chief conclusion that in the case of the pneumococcus, the bacillus pyocyaneus, and the diphtheria bacillus the weak, irregular, and arrested circulation which has hitherto been attributed to heart failure is due to paralysis of the vaso-motors. The heart does show a characteristic picture from a clinical standpoint, but the weakness of the heart is entirely overshadowed by the paralysis of the vaso-motors; moreover, the various infections show a remarkable unanimity of action, in that it is always a paralysis of the centre in the medulla which produces the disturbance in the circulation (Romberg and Bruhns).

Thus, arrhythmia may be due to direct, reflex, or toxic cause, or to the effect of two or all of these causes combined.

The cause is *direct* when it proceeds from some lesion in the brain or spinal cord which implicates directly or indirectly the centres of the pneumogastric and accelerator nerves. Thus, arrhythmia is observed in meningitis, and is sometimes a valuable index in diagnosis, and more especially in prognosis, in this disease. Cerebral hemorrhage, apoplexy from any cause, is more apt to show a transitory arrhythmia as the result of indirect pressure upon the nerve centres. Organic lesions, brain tumor, abscess, etc., which directly involve the origin of the nerves, may show a more persistent arrhythmia. Pressure upon the nerve trunks, as by enlarged glands, neoplasms in the neck or in the mediastinum, hypertrophy of the thyroid and thymus glands, aneurysm at the base of the brain, or, more especially, at the arch of the aorta, may have the same effect.

Arrhythmia may be observed also in connection with various organic diseases of the heart itself, as in the course of peri- and endocarditis, myocarditis, arterio-sclerosis, etc. It is easy to understand how an insufficient blood supply to the brain, under which head may be included alterations, especially degradations, in the blood itself, as in hemorrhage, anæmia, chlorosis, leucocythæmia, may act in the same way. It is easy to understand also how distention of the stomach or large intestine with gas, or upward displacement of the diaphragm under the pressure of fluid, may mechanically interfere with the action of the heart and disturb the regularity of its rhythm. Typical arrhythmia may be observed in the preagonal period, as in nearly all cases the heart action becomes irregular as well as faint before it ceases altogether.

In the development of idiopathic hypertrophy, as produced either by arterio-sclerosis in the peripheral vessels or by the spastic contractions caused by the presence of poisons in Bright's disease, arrhythmia is a sign of much significance. The patient himself becomes conscious of the interrupted or intermittent action of the heart, noticing the condition first or more especially after a full meal or after some mental emotion, so that the disturbance in the rhythm of the heart, associated often with attacks of palpitation, is an index of the condition of the vessels.

Reflex irritations may reach the heart from any organ in the body, but are most frequently observed after affections of the abdominal organs. A sudden shock, as from extensive trauma, contusion, or crushing of great organs, may arrest the action of the heart. Pain may be so severe as to stop its beat. Extensive injuries to the kidneys or uterus may have this effect, and the connection of the nervous system is evidenced in the experiments of Brown-Séquard, who found that extirpation of the semilunar ganglia arrested the action of the heart, but if the vagus had been previously cut, so that the nervous impression could not be transmitted, the heart continued to beat as before. The same protection to the nerve centres is secured in the administration of anæsthetics before an extensive operation.

The *toxic* causes are represented especially in the action of the poisons developed by the infectious diseases, which act indirectly upon the muscle or the nerves of the heart. The toxins of various infections, especially those of graver character, such as typhus fever, scarlatina, cerebro-spinal meningitis, cerebral rheumatism, and diphtheria, indirectly poison the muscle of the heart. Illustration of this poisonous influence is furnished in the action of muscarin, which at first retards the heart and later renders it irregular and arrhythmic. Digitalis, strophanthus, and various arrow-poisons, chloroform, and chloral may act in the same way. Tobacco exercises its effect also indirectly upon the nerves of the heart. Nicotin at first in small dose retards the pulsations, but in large dose it may arrest them entirely. In the course of time the action of the heart becomes irregular and finally faint and imperceptible. That this action depends upon irritation of the peripheric filaments is proven by the fact that after the injection of curare and atropine, substances which paralyze these filaments, the peculiar effect of tobacco is not observed at all. The frequency of arrhythmia under the use of tobacco is established in the observations of DeCaisne, who found among 88 smokers intermittent action of the heart twenty-four times. Richardson remarked upon the intensification of an intermittent action of the pulse after the use of tobacco, and Liebermeister observed that the smoking of but one or two cigars a day would suffice to sustain the arrhythmia which had been once produced under greater excess.

Coffee and tea gently stimulate the heart, but in abuse exhaust it.

Alcohol is an illustration of combined effects, for alcohol acts in the first place as a toxin, which in large dose expends its force upon the nervous system, and produces the well known symptoms of intoxication with derangement in the action of the heart. Under more protracted use alcohol begets the changes of arterio-sclerosis, and thus by increasing the resistance of the vessels throws extra work upon the heart, and in the course of time wears it out under quicker and more irregular action. Alcohol is the most frequent of all single causes of arrhythmia. So much is this the case that the practitioner should first of all eliminate alcoholism as a factor in any disturbance in the rhythm of the heart.

An irregular pulse in children has always been considered pathognomonic of tuberculous meningitis. Heubner found it, however, in a series of other diseases and conditions, as in poisonings by stramonium; in auto-intoxication from the intestine; in diseases of the abdomen connected with vomiting; in the beginning of convalescence from acute

infections. Arrhythmia has also been observed in anæmic and nervous children between two and four years of age, after puberty in thin and rapidly growing individuals, in irritation from intestinal parasites (Bouchut, Barthez, Saunét), under certain physiological conditions (after mental excitement, in sleep, after warm baths, in the subsequent cooling), and as a condition *sui generis*, constituting the so called "idiopathic" arrhythmia.

SYMPTOMS.—The term itself indicates a symptom, and the arrhythmia shows itself, as the name implies, in disturbance of regularity in the action of the heart. This irregularity may consist, as stated, of a simple intermission, and the intermission may occur irregularly or may be itself regular with every sixth, tenth, or twelfth beat. In other cases the irregularity consists in a reduplication of beats or in the suppression in force of individual contractions. Beats at short intervals and at long intervals, beats that are tumultuous and bounding or under feeble and faint contractions, constitute different forms.

Where the arrhythmia is slight the patient may be entirely unconscious of the condition, which is then first observed, for the most part, usually by the physician. In other cases the patient is aware of the condition or becomes painfully apprehensive of danger under the irregular action of the heart. Certain patients speak of feeling the trip or jerk, and experience at times choking sensations, or they may be threatened with vertigo or syncope. For the most part, the interruption when perceived is attended with no real distress, but sometimes there is positive pain, which may at times, with an associate sense of danger, become so severe as to approach the excruciating tortures of angina pectoris.

Interruption or irregularity of whatever character announces itself at first under some emotional disturbance or after severe bodily strain or mental effort, still more frequently after the ingestion of a full meal when the bloodvessels are overloaded and extra work is thrown upon the heart.

Sometimes arrhythmia is the sole sign of disturbed action. In other cases there may be, besides, irregular action, palpitation, or the pulsation of the heart may be increased or decreased to such degree as to constitute tachycardia or bradycardia.

Other symptoms which may be associated with the condition belong to other affections or are the result of some common cause. Thus, the presence of dyspnœa would indicate a myocarditis or a thrombotic occlusion of branches of the pulmonary artery; cyanosis would depend upon the same cause, and dropsy might be the result of chronic valvular disease. Attacks of arrhythmia which may occur in the course of tabes dorsalis constitute the so called heart crises. Like the similar explosions in the larynx, stomach, and kidneys, these attacks occur suddenly without evident exciting cause, and are attended with pain, anxiety, and sometimes syncope.

Buchholz encounters among the hysterical a vagus neurosis which shows prominent symptoms on the part of the circulatory apparatus, the throat, and intestinal canal. Many of the cases are introduced by analgesia, aphonia, convulsions, globus, and clavus, but the most remarkable symptom is the arrhythmia, which sets in without any outside

cause and is distinguished by its rapid change. The pulse frequency is usually somewhat increased. Sometimes there is a typical tachycardia. The arrhythmia which shows itself at times in the course of neurasthenia has something of the same history, save that the overshadowing symptom is weakness of the whole body, with anxiety, insomnia, anorexia, etc., sometimes with a history of excesses or perversions in the sexual sphere.

Diagnosis does not enter into consideration, as the condition reveals itself upon simple examination, and questions in diagnosis hinge wholly upon the cause of the condition.

Prognosis also takes color from the cause. In a general way it may be said that arrhythmia is a sign of some significance. It is true that an arrhythmia of light degree is natural with some people. It is sometimes noticed in children, especially in sleep, disappearing on awakening. An arrhythmia which is regular—that is, an allorrhythmia—need not necessarily be of grave import. The arrhythmia which is an expression of indigestion, as in distention of the stomach, or may result from accumulation of new matter in the blood, has no serious significance. On the other hand, the arrhythmia which indicates implication of the heart in the course of Bright's disease, lead-poisoning, or gout may signify the beginning changes of arterio-sclerosis, a very serious disorder. The arrhythmia which results from the action of toxins in the course of infections like diphtheria and scarlet fever is very grave, as this symptom is sometimes observed several hours or days before death. But the arrhythmia which results from the ingestion of irritants, such as alcohol, coffee, and tea, may disappear completely with cessation of their use.

As a general rule, it may be said that arrhythmia is more grave than palpitation or than tachycardia, though it is not so serious as bradycardia, and is far less ominous than angina. An arrhythmia which is the expression of heart failure is always grave. The arrhythmia which may establish itself during sedentary life with irregular habits, under an increase in the amount of blood, may disappear entirely with exercise and regulation of life. An arrhythmia which disappears under exercise has little or no gravity, but an arrhythmia which is intensified by it may indicate heart failure from defective nutrition, from coronary sclerosis or cicatricial myocarditis. An arrhythmia the result of exhaustion or of hemorrhage may entirely disappear under rest and restoration of the natural amount of blood.

Treatment.—The treatment must be addressed wholly to the cause. Bad habits, abuse of stimulants—including under this head tea, coffee, and tobacco—night vigils must be abandoned. Sources of anxiety may be removed. The arrhythmia which depends upon the accumulation of toxins in the course of gout, lead-poisoning, and Bright's disease may be relieved by the treatment of these various affections. A simple arrhythmia which in no way disturbs the health of the patient should be let alone. The arrhythmia which is the expression of heart weakness may be relieved by gentle stimulation of the heart, especially with strychnine or the tincture of nux vomica, and more especially by regular graded exercise, which tones the muscle of the heart. Appeal to the stronger stimulants, digitalis, strophanthus, may be made in relief

of the associate weakness, and the nitrites may be administered in the presence of pain. Extreme failure may call for the use of camphor and caffeine, which may bridge over a threatening collapse and protract life indefinitely notwithstanding the arrhythmia. Irregularity which is due to hemorrhage or which may occur in the course of exhausting discharges may be sometimes relieved by the subcutaneous injection of a half to one pint of the physiological salt solution, 0.6 per cent. The irregularity of arterio-sclerosis requires longer treatment with regulation of the habits, which must be made temperate and more abstemious in every way, with the avoidance on the one hand of too much sleep, and on the other of exercise up to or beyond the point of fatigue.

The arrhythmia of depressing mental emotions, homesickness, love-sickness, disappointment, domestic infelicities may be cured only by relief of the cause.

Palpitation.

Palpitation is the beating of the heart which is felt as a distress by the patient. The heart may beat too fast or too slow, too strong or too weak, but none of these factors alone or conjoined constitute palpitation. Ordinarily, the pulsation of the heart is not perceived. It is only when called upon to produce extra demands, as after physical exercise or under the influence of some emotional strain, that the heart beats are felt; and then the heart beat may be not only felt, but heard by the patient, and may be perceived by others in the vicinity. This degree of palpitation is physiological. The heart beats with additional force to supply extra demands, or the heart is suppressed by a shock and the palpitation is the effort at adjustment under the increase or decrease of stimulation. Palpitation is pathological when it occurs without known cause or when the response of the heart is out of all proportion to the demand.

The mechanism of palpitation is difficult of comprehension. The heart is exceedingly sensitive and the nervous apparatus is as yet inextricably complex. It is difficult, therefore, to determine whether the disturbed action of the heart is due to arrest of inhibition (vagus) or disturbance in the accelerating (sympathetic) functions. It is admitted that the action of the heart may be increased or disturbed in any way by affection of the heart muscle independent of any nervous influence. Palpitation is often divided into that which depends upon organic and that which is assumed to be of functional cause. The palpitations whose causes lie in the nervous system or are reflected through the nervous system constitute the so called nervous palpitations.

Etiology.—For clinical purpose palpitations are found to depend upon or to be produced by direct, reflex, or toxic cause. Palpitation is said to be *direct* when it depends upon some organic disease of the heart itself. These palpitations are found most frequently in the so called idiopathic hypertrophies, and especially in the cases dependent upon arterio-sclerosis in which the heart is deprived of the assistance of the bloodvessels, and must do, as it were, double duty. The enlargement of the heart which occurs in consequence of the spastic contraction of the vessels induced by Bright's disease is likewise announced by a preliminary stage of palpitation.

Under direct causes are included also irritations in the brain, which may involve directly or indirectly the heart nerve centres in the brain and cord. The palpitations which occur under the emotions result from inundations of nerve force from the cerebrum upon the heart nerve centres at the base of the brain. Palpitations are observed also in the course of organic affections of the central nervous system, involving the nuclei of origin of the heart nerves. Thus the heart crises of tabes dorsalis are caused by sclerotic changes in the medulla. Palpitation occurs also in the course of disseminated sclerosis, progressive paralysis, and not infrequently as the precursor, sometimes as the actual aura, of epilepsy or migraine. Palpitation of the heart is one of the triad of symptoms which indicate the onset of Basedow's disease, and the occurrence of palpitation may indicate in a case of lateral or posterior sclerosis such implication of the medulla as develops later a true bulbar paralysis. Hysterical and neurasthenic patients furnish a large contingent of cases of nervous palpitation.

Irritation of the heart nerves in their course in the neck may have the same effect. Thus cases of palpitation have been found to be due to ganglion swellings upon the vagus nerve (Proebsting) or to pressure upon the nerve by caseous tuberculous glands (Riegel, Pelizäus). The nitrites, especially amyl nitrite, produce palpitation by paralyzing the terminal filaments of the vagus in the heart and thus cutting out the inhibitory influence of the nerve.

Much more numerous are the *reflex* causes in which some irritation is propagated from a distant organ, especially from the organs in the abdomen and pelvis. Palpitation is a common sign of affection of the stomach. Sometimes it is due to mechanical interference with the action of the heart under distention of the stomach or colon. More frequently the influence is toxic and results from the absorption of toxins. Dyspeptics all suffer attacks of palpitation of the heart. Palpitation occurs also in the course of obstruction in the portal system, and may be evoked or aggravated by simple constipation. Affections in the genito-urinary sphere are followed by palpitation, as a rule. Certain cases are relieved by a proper treatment of prostatitis, salpingitis, etc. Sexual excesses, more especially unnatural indulgence and perverted relations, are attended by palpitation, as a rule. There is close relation between the heart and the genital sphere, and the changes initiated by puberty are marked by irregularities in the action of the heart. Attacks of palpitation, which may thus occur at any time, are excited or aggravated by bad habits, by sexual indulgence, perversion, or excess, or especially by masturbation with its moral degradations. Fothergill reported an obstinate case in which the cause was found to depend upon irritation in the rectum, the removal of which prevented the attacks. As stated elsewhere, extreme pain may paralyze the heart or may, by arrest of inhibitory influence, lead to the disturbance of palpitation.

The *toxic* causes are agents which directly poison the heart muscle or disturb the circulation through the nerve centres. Common causes are the heart stimulants, alcohol, tobacco, coffee, and tea. Any excess in the use of these agents is liable to be followed by attacks of palpitation, and when the condition has once been established the slightest indulgence precipitates attacks. Bright's disease causes palpitation in

two ways—by throwing extra work upon the heart and by deranging the circulation through the action of toxins. Gout acts in the same way. Gout is a frequent cause of arterio-sclerosis, and the blood is loaded by it with toxic matters.

Finally, palpitation may be produced by degradations of the blood itself. The various forms of anæmia, leucocythæmia, sclerosis, scurvy, etc., including under this caption the loss of blood by hemorrhage or impoverishment from any exhaustive discharge, are marked by attacks of palpitation. The term *erethismus cordis* has long been applied to indicate the easy excitement of the heart in the inception or during the course of *tuberculosis pulmonum*. Palpitation shows itself also in the course of inanition or marasmus from any cause.

On the other hand, over-accumulation of blood which distends the bloodvessels evokes attacks of palpitation. Something akin to this condition is observed in every individual after a full meal, and the high liver soon experiences palpitation as the first indication of plethora. Here too, however, may be observed the influence of conjoint cause, as the palpitation which sets in after a full meal has been attributed to the overloading of the blood with peptones.

Symptoms.—Palpitation is itself a symptom, and the beating of the heart is appreciated by the patient as such an obvious distress as to make itself distinctly manifest. Palpitation varies with every degree of intensity. Sometimes it is so slight as to excite only a little discomfort, and in no way interrupt the avocation or pursuit of the individual. In other cases the palpitation is violent. The heart beats with force against the wall of the chest, which is visibly agitated under its stroke. The patient feels as if the heart would burst its bounds. In these cases the face may be flushed and anxious, the eyes suffused, and the vessels throb in the neck. The respiration is deep and sighing: there are ringing in the ears, sparks before the eyes, vertigo, with tremor and agitation of the whole body. The patient is now unable to speak. He sits or lies with his hands upon the heart, wearing an expression of anxiety, and whispers or articulates his wants or sufferings in broken words. The face is pallid and may be covered with a clammy sweat, the extremities are cold, the lips may be blue, and in extreme cases there is a tendency to syncope. Lighter cases are attended with eructations and borborygmi, and are often relieved by the free discharge of gas.

The attacks occur, for the most part, in the daytime, and are excited by some trivial cause or set in suddenly without any apparent cause. They are often provoked by emotional excitement or occur immediately after the taking of a glass of wine or a cup of coffee or tea: a sudden shock of any kind may precipitate a violent attack, and apprehension or a state of suspense, as the mere awaiting an engagement, especially if it be deferred, brings on an attack. More infrequently palpitations occur in the night and awaken the patient from a sound sleep with a feeling of great anxiety. Nocturnal attacks should always excite the suspicion of epilepsy. Sometimes patients are more alarmed by the subsidence of the beating than by the attack. They have no fear, they say, so long as the heart beats, but are frightened when the heart apparently ceases to beat, so that they may lie awake in the night anxiously awaiting a perceptible restoration of the action of the heart.

Physical Signs.—Aside from organic disease, of which the palpitation may be only a symptom, the heart may show no anomalies. The outlines are only apparently enlarged, and the greater agitation of the chest is due simply to the increased impact of closer and more extensive contact of the heart with the walls of the chest. The sounds of the heart are all intensified, to show often under tumultuous action metallic resonance. But there is no anomaly in the action of the valves, though they are shut with such force as to be intensely accentuated—*cliquétis metalliques.*

The attack lasts for a few minutes to the greater part of an hour or more, or sometimes recurs at various times during the day. The purely nervous palpitation often subsides under a profuse sweat or free action of the kidneys.

Diagnosis is, for the most part, simple. There is no doubt as to the existence of palpitation and the question of interest in every case turns upon the cause—that is, whether the palpitation is of purely nervous origin or is a symptom of organic disease. The diagnosis in this case is reached by exclusion. It is observed in organic disease that the diameters of the heart are increased, that murmurs are present, or that individual sounds are distinctly accentuated. The purely nervous palpitations are found most frequently in cases of neurasthenia and hysteria or in connection with bad habits, including masturbation, sexual perversions, night-vigils, hemorrhage, exhausting discharges, etc. Purely nervous attacks are found in association with or provoked by disturbance on the part of the nervous system by shock, anxiety, worry, grief, including home-sickness, love-sickness, disappointment, etc.

Prognosis depends upon the meaning of the symptom, whether it expresses a purely nervous condition (neurasthenia), impoverishment (anæmia), or is the evidence of some organic diseases (myocarditis). A purely nervous palpitation, however severe, does not take life and does not entail organic disease, though the cause upon which it depends, some reflex irritation, may finally exhaust the heart. Thus a long continued hemorrhage will lead to a fatty degeneration, or the toxin of some infection (prostatitis), which may produce only palpitation at first, may finally develop a myocarditis.

Treatment.—The treatment resolves itself into the treatment of the attack and treatment of the intervals. During the attack the patient should remain as quiet as possible, and should lie in bed or should observe the semi-recumbent posture. All anxiety must be, as far as possible, allayed. The assurance of the physician that the malady is not organic, that it is temporary and remediable, is an important element in therapy. The attack usually dies away under absolute rest in the course of a few minutes. In the more aggravated case the chest may be bared and the windows opened that more perfect ventilation may be secured, or the patient may find relief in the process of fanning the surface of the chest. The average attack may be cut short by the administration of a teaspoonful of good French brandy, or by a half to one teaspoonful of Hoffmann's anodyne, or the compound spirits of ether, or the aromatic spirits of ammonia. Excessively tumultuous action may be stilled by the application of an ice bag or by cloths wrung out of cold water applied freely to the chest. Milder

cases may be relieved by the use of a salt of caffeine, especially the sodium benzoate, in dose of 3 to 5 grains, which is best administered in a glass of effervescent (Seltzer) water. Sodium bromide in dose of xx–xl grains, administered in the same way, has a more permanent effect. Valerian is always a simple and safe remedy for mild cases; for chronic cases Rosenbach recommends ergotin in pill of 1 or 2 grains, three to six per day. Obstinate cases call for the use of morphine, best given subcutaneously in the dose of ¼ grain.

Attention is next directed to the discovery and removal of the cause, which is sometimes found in disease of some distant organ. The treatment may here have reference to the relief of Bright's disease by hot baths, tuberculosis by creasote and tuberculin, prostatitis by the use of deep injections of lactate of silver, salpingitis and endometritis by appropriate operative procedures, to regulation of diet, to exercise in the open air in hypertrophy from spastic contraction of the peripheral vessels, etc. etc.

In the purely nervous cases bad habits are to be surrendered and all cardiac stimulants, including alcohol, tobacco, coffee, and tea, abandoned altogether. The digestion may be improved by the regulation of the diet with the use of dilute hydrochloric acid and of bitter tonics, and constipation overcome by the administration of suitable laxatives—Carlsbad salts, cascara, rhubarb, podophyllin. The anæmia which results from hemorrhage or exhausting discharge is combated best after relief of the cause by the use of the preparations of iron and arsenic. A happy combination of these agents is found in certain mineral waters, in the Roncegno and Levico mineral waters, of which a dessertspoonful to a tablespoonful may be given in a wineglassful of water after meals. Neurasthenia and hysteria require additional psychical treatment. A change of climate, mountain or sea, may be necessary to break up a bad case.

Tachycardia.

Tachycardia (ταχύς, quick) is the name used to denote a disturbance in the action of the heart which is expressed in increased frequency. The name is new, but the condition has long been recognized and described under palpitation. It was the observation of the fact that the increase in frequency sometimes occurred in attacks which gave acceptance to the term, and the periodical increase was designated as paroxysmal tachycardia. As already stated, palpitation is distinct as a beating of the heart which is felt by the patient. The increased frequency which constitutes a tachycardia may be attended with palpitation and may be appreciated by the patient, but not rarely the increase is recognized only by the physician. The patient is usually conscious, however, of other signs of distress.

Mere increase in frequency does not constitute tachycardia. Increase in frequency is natural with some people, and occasional increase to 100 to 120 results often from increase in muscular exercise or from emotional strain. Moreover, the pulse is increased in most fevers, during convalescence, and in states of exhaustion as the effort of the weakened heart to adjust itself to the increased demands made upon it. Thus, tachycardia may be permanent, periodic, or paroxysmal in occurrence.

Permanent tachycardia, aside from the rare cases in which the condition is natural, is usually an expression of some grave affection of the nerve centres. Perhaps the most common cause of permanent tachycardia is Basedow's disease, which is supposed to depend upon some lesion of the nerve centres or some irritation of these centres by toxins. A rapid increase of frequency in the action of the heart, associated with attacks of palpitation which are evoked by the slightest effort or nervous impression, or which occur at times without discoverable cause, should lead, especially in a nervous subject, to investigation for the other symptoms of the disease, especially tremor, exophthalmos, and goitre. Permanent tachycardia is often the expression of weakness in a heart which has been enfeebled by fatty degeneration or cicatricial myocarditis.

Periodic or transitory tachycardia is found, as stated, mostly in connection with fevers, especially of the infections, during convalescence, and in states of exhaustion from loss of blood, anæmia, etc. Inundation of the blood from time to time by toxins which accumulate under the action of micro-organisms or their products excite attacks of tachycardia. The heart is increased in frequency preceding or during the invasion of new joints in rheumatism or in connection with new localizations of sepsis.

Paroxysmal tachycardia, as the name implies, is represented in attacks which occur at certain indefinite periods, with normal pulsation in the intervals. The condition would seem to be caused by arrest of the inhibitory influence of the vagus, which allows the heart to run away with itself, as Balfour puts it, like the hands of a clock from which the weights have been removed. This paroxysmal tachycardia may be the expression of disease in the nerve centres which operates only from time to time as they may be irritated, especially under the accumulation of toxins. The elimination of the toxins stops the attack.

Etiology.—Tachycardia may result from direct, reflex, or toxic causes. Direct causes are those which operate chiefly upon the pneumogastric nerve, either in the nerve centres in the course of the trunk or in the nerve filaments in the heart. The attacks of tachycardia which occur in emotional strain, under violent shock, fright, anxiety, joy, etc. express the withdrawal of the inhibitory force of the cerebrum. Hysterical, hypochondriacal, and neurasthenic patients suffer frequent attacks of tachycardia with or without palpitation. Organic affections of the brain, meningitis, sclerosis, tumors, softening, may have the same effect. Typical attacks have been observed to occur in consequence of tumors in the neck, caseous glands, affections of the mediastinum, etc. The attacks of increased frequency which develop in connection with affection or degeneration of the heart muscle itself fall also under the head of the direct causes.

Reflex causes operate upon the nerve centres from distant organs, though the disturbed action from this cause is represented more commonly in palpitation than in tachycardia.

Toxic causes are represented in the increased frequency which occurs in connection with Bright's disease, tuberculosis, rheumatism, and occasionally in gout and lead-poisoning—diseases which oftener retard than accelerate the action of the heart.

Paroxysmal tachycardia, when of nervous origin, is supposed to be

due, as stated, to the irritation of the nerve centres under the accumulation of toxins, often of cryptogenetic source, or is ascribed to molecular changes in the nerve centres of the same nature as those which are assumed to exist in epilepsy and migraine. Clinicians are inclined to adopt one or other of these views according to bias in favor of morphotic or chemical cause. When paroxysmal tachycardia occurs more distinctly in connection with organic disease, it is usually attributed to degeneration, especially to dilatation of the heart. Martius especially maintains this view, ascribing all bad cases to an acute dilatation, on the ground that an organic lesion of the brain may not produce temporary or paroxysmal effects.

Symptoms.—Tachycardia, as the name implies, is an increase in frequency of the pulse, and to constitute a disease the increase must occur in paroxysms unprovoked or excited only by trivial cause. Just what must be the degree of frequency to constitute tachycardia is nowhere definitely stated, but it is recognized that the pulse rate is increased to 140, 160, 200, and more. In fact, in most cases the pulse is so rapid that it may be counted only in fractions of a second, so that the rate for the minute is estimated by multiplying the beats in proper ratio.

Paroxysmal tachycardia sets in for the most part suddenly and in the midst of apparent health. That is, the individual enjoys the amount of health which is usual in the neuroses. As in epilepsy, migraine, etc., there is a substratum of disease which is represented in an unstable condition of the nerve cells, and this condition may make itself manifest in changes of disposition, irritability, emotional nature, etc. These fine changes pass, however, for the most part unnoticed, and the attack sets in, as stated, suddenly and without premonition. The heart now begins to beat with increased rapidity and the pulse runs up from 60 to 80, the natural frequency, to 140, 160, 200, and more.

The rapid increase in rate of the pulse is attended with distinct disturbance in the nervous system. Cases vary in every degree of severity, but in an average case there are apprehension and anxiety. The patient is unable to stand up or lie down, but sits in an upright or semi-recumbent posture, prostrated with anxiety, unable to speak save in whispers or broken words, while the face may be bedewed with a cold sweat. Palpitation may or may not be present. Sometimes, as mentioned, the disturbance on the part of the heart is not appreciated by the patient, who suffers only a general anxiety and distress, occasionally with a sense of imminent danger. The condition is disclosed by an examination of the pulse, which beats so fast, as stated, that it may be counted only in the fraction of a second. Vibrations too rapid to be recognized by the finger may be detected by the sphygmograph.

In a pure case of paroxysmal tachycardia the heart shows no evidence of organic lesion. Where the pulsation is very rapid it is difficult to eliminate the various heart sounds, which seem to run together; or, if they may be separated, they show themselves of uniform intensity, thus imitating the condition of the embryo heart. The existence of a murmur or the evidence of enlargement of the diameters would indicate some coincident organic disease, or the presence of any positive pain would refer to a complication with neuralgia, or possibly with calcification of the coronary arteries (angina pectoris).

The symptomatology is thus summed up as a rapid increase in the action of the heart, with general distress and anxiety. The breathing is but little affected, but is sometimes sighing. Anything like distinct dyspnœa would indicate muscular failure of the heart or complication on the part of the lungs, especially œdema of the lungs from heart failure. The attack lasts usually the greater part of an hour, but sometimes extends over the greater part of a day, or, with exacerbations and remissions, persists for several days. Bristowe reported a case which lasted for five weeks. It is in these protracted cases that the frequency of the heart is apt to be most increased, at times up to 260, and even up to 300.

The attack ceases as suddenly as it set in, and the end of the seizure is usually announced by the inundation of the arteries by a great tidal wave, which is followed by a number of smaller waves, after which the action of the heart is restored to its natural rate and force. The color now returns to the face with a feeling of warmth over the whole body; the nervous dejection and anxiety disappear. The patient sits up and resumes his work, and is restored to his natural state. As in the case of other neuroses, the end of the attack may be attended by a profuse discharge of urine or profuse sweat or diarrhœa.

Attacks may repeat themselves frequently or only at long intervals. Sometimes they occur in a series of paroxysms. Rosenfeld recorded the case of a patient who was supposed to have entirely recovered, but in whom a subsequent attack occurred after the lapse of fifteen years.

Diagnosis.—As tachycardia is only a symptom, the diagnosis which depends upon the recognition of the symptom is easy enough. To constitute the disease in question the condition must occur in paroxysms, with intervals of normal action of varying length. The attacks set in, as stated, suddenly, and are attended with nervous phenomena—with anxiety, sometimes with vertigo and vomiting, and with a sense of faintness and prostration. They terminate suddenly, sometimes, after the manner of other neuroses, in diuresis, diaphoresis, diarrhœa, etc. After the attack the patient feels as well as before, or is left with a feeling of languor, or falls into his natural state of neurasthenia.

Prognosis.—The prognosis is favorable so far as life is concerned. The condition itself, however, is very obstinate and defiant of relief, as it probably depends either upon some organic molecular lesion of the nerve centres or upon marked degeneration of the muscle of the heart itself. That most cases owe their origin to nervous disturbance, and not to organic disease of the muscle, is manifest in the fact that the extreme, almost incredible, increase in frequency does not impair the integrity of the heart, which during the intervals beats with normal force and frequency. Nevertheless, where the disease has lasted for a long time it may finally beget organic changes, and it is not surprising to learn that Bouveret was able to find in the literature four cases of sudden death from heart failure during an attack. But the localization of the origin in the nervous system by no means makes the prognosis trivial, as it is generally conceded that the lesion of the nerves implies some, however slight, change of structure.

Treatment.—The treatment of tachycardia is addressed to the relief of the attack and to the toning of the nervous system and of the

heart muscle during the intervals. The treatment of the attack is based upon the inhibition exercised by the pneumogastric nerve. It has long been recognized that compression of the vagus in the neck retards the action of the heart, and may in this way arrest an attack of tachycardia. Bensel and Weidener succeeded in aborting an attack within two to five minutes by mechanical irritation of the vagus in the neck, and cases treated with similar success were reported by Czermak and Priesendörfer. These patients had learned themselves to irritate the filaments of the pneumogastric nerve, without any knowledge of the nature of the process, by simply holding the breath for a long time. Nothnagel knew a patient who could cut short an attack by a full and deep inspiration, and in a case recorded by Rosenfeld the patient adopted this procedure as a regular means of aborting an attack. This patient, a lady, so soon as she was seized, stretched herself out in bed, raised the head, and pushed the feet against the foot of the bed; hereupon she took a deep breath and subjected the lungs to the greatest degree of compression by contracting the abdominal muscles and holding the breath. The chest was fixed laterally by pressure of the arms, and was held immobile in this position for fifteen or twenty or more seconds. Under this irritation the inhibitory power of the vagus is exercised to the utmost degree, and attacks of tachycardia are arrested at once. During the maintenance of the compression the vessels throb in the neck, the eyes bulge, and the face, lips, and nose are cyanotic. These effects disappear, however, so soon as the attacks subside, and the method, though it is not always attended with success, may be recommended as a safe procedure in similar cases.

This contribution of therapy is all the more welcome from the fact that the agents of materia medica have over the condition little or no control. Thus, morphine and atropine may sometimes, but they seldom, succeed in cutting short an attack. Occasionally cases are relieved by the inhalation of amyl nitrite, and nitro-glycerin has been recommended empirically.

During the intervals the heart muscle may be strengthened by graded exercise in the open air, and the nervous system may be supported by hydrotherapy, electricity, and massage. The virtue of strychnine, especially in subcutaneous use, is well established in sustaining the action of the heart by heightening the reflexes and thus furnishing additional nerve force. Arsenic is always of value in improving assimilation. All bad habits must be broken up. Abundance of sleep must be secured, and sources of anxiety as far as possible relieved. All reflex irritations must be removed. Illustrative in this connection is a case reported by Watson of a man aged thirty-five, who had suffered for four months from increasing obstruction of the nose, which had in the last part of the time been attended by a disagreeable tachycardia and pains in the region of the heart. Examination showed that both sides of the nose were nearly occluded by polypi. After removal of this obstruction the tachycardia disappeared permanently.

Conditions of anæmia, chlorosis, gout, etc. will call for especial treatment. The same thing may be said of neurasthenia and hysteria, associated underlying conditions in the development of tachycardia.

Bradycardia.

In bradycardia (βραδύς, slow) the action of the heart is retarded. The normal pulse ranges between 60 and 80 per minute, but excesses in either direction may not be incompatible with health. The pulse in some people is naturally fast, in others naturally slow. Dunglison declared that the pulse of Sir William Congreve beat 120 times per minute, and quotes, on the authority of Cruveilhier, the statement that the pulse of Napoleon never exceeded 40 beats per minute. Slow pulse is often found in self-possessed and unexcitable people, and sometimes it runs in families. It is necessary in all cases to appreciate the fact that the action of the heart and the pulse do not always correspond. Sometimes the pulse is apparently slow, from the fact that all the pulsations of the heart do not reach the artery.

How slow may the pulse be to constitute a bradycardia? The authorities differ upon this point, but it is usually conceded that bradycardia begins only with a reduction to at least 40 beats per minute.

Bradycardia is by no means as frequent as tachycardia. Thus Grob found but 32 cases in 578 observations, Rosenbach but 4 in 100 observations. The male sex preponderates. Grob's cases were all men but 4, and but 6 of the 100 cases recorded by Rosenbach were women.

Bradycardia is permanent, temporary, and paroxysmal.

Permanent bradycardia is usually an expression of organic disease of the brain. The pulse is reduced to 50 or less in states of stupor, even in the absence of changes in the walls of the arteries. Permanent bradycardia may develop in any case in which the pneumogastric nerve is directly or indirectly irritated in the brain; thus in the course of pyelitis, pachymeningitis, cerebro-spinal meningitis, and from trauma. Gurlt found the pulse reduced as low as 20 per minute in a case of injury to the seventh cervical vertebra, and Charcot accepts the retardation of the pulse as a symptom of lesion of the spinal cord in the neck. Hutchinson holds that an injury is without influence upon the action of the heart unless it occurs in this region. Hydrocephalus, tumors, and abscesses may have the same effect. Epileptics are often found to have a slow pulse. Retardation of pulse in these cases is clearly the effect of a common cause.

Temporary bradycardia is much more common, and is found in the course of various infections, especially after typhoid fever and diphtheria, pneumonia, erysipelas, and rheumatism. Bradycardia occurs in about 25 per cent. of cases of puerperal fever. Baumgarten found the pulse reduced to 25 per minute late in the course of diphtheria. The accumulation of bile salts in the blood retards the action of the heart, so that a slow pulse is found in jaundice. The absorption of toxins in the alimentary canal may have the same effect, and the slow pulse which is often seen in the course of occlusion of the intestine stands in vivid contrast to the viciousness of the signs.

Paroxysmal bradycardia sets in suddenly in the midst of apparent health, and subsides, as a rule, along with other manifestations of great distress on the part of the nervous system. Bradycardia may arise from direct, reflex, or toxic cause, or, as in the case of other neuroses, two or all these causes may be combined in an individual case.

Bradycardia is of direct origin when it depends upon disease or injury of the brain and cord or of the trunk or filaments of the vagus nerve in its course. Thus, Wreden reported a case of retardation to 10 in the minute in the case of a soldier the subject of abscess of the brain. Jacobi saw a case of epilepsy in which the pulse was reduced to 7 beats per minute, and retardations as great as this have been frequently recorded during the sleep attacks of hysteria.

Irritation of the trunk of the vagus nerve in the neck may thus also arrest the action of the heart. Czermak could stop his own heart for several beats by compression of the vagus nerve against an exostosis from one of his cervical vertebræ. Concato verified this observation once upon a patient.

Etiology.—Bradycardia is due to direct, reflex, and toxic cause.

Direct cause is found, as stated, in disease of the brain, in irritation of the vagus at its origin, course, or termination in the heart.

Reflex bradycardia arises for the most part from some irritation in the abdomen. Kisch sometimes saw marked retardation during the rest-cure with overfeeding. Bradycardia from uræmia is occasionally encountered in the course of Bright's disease. In this case the chemical is conjoined with the reflex cause.

Toxic bradycardia occurs thus under the influence of uræmia, icterus, and various toxins. The modus operandi in these cases is illustrated in the action of muscarin, which enormously retards the pulse and finally stops the heart altogether. Chronic cases find their best exemplification in lead-poisoning and gout.

Sometimes the irritation of the vagus which stops the heart arises in the heart itself, but valvular lesions are seldom attended with slow pulse. Retardation of the pulse occurs in the course of myocarditis, but only in exceptional cases. Thus, Quain found a slow pulse in only 8 of 51 cases of degeneration of the substance of the heart. Bradycardia from heart disease is most typically shown in the case of angina pectoris, where it is dependent upon calcification or occlusion of the coronary arteries. Retardation of circulation in the heart reduces the frequency in the action of the heart, and organic disease, especially cicatricial myocarditis, may be marked by distinct bradycardia.

The ratio of causes was established by Prentiss from his analysis of 91 cases found in the Index Catalogue of the Library of the U. S. Surgeon-General's Office. The cause of the bradycardia was supposed to be in these cases disease of the brain 8 times, of the cervical vertebræ 11 times, epileptic convulsions 7 times, heart disease 9 times, calcification of the aortic valves and coronary arteries 3 times, starvation and exhaustion 3 times, lead-poisoning, nervous shock, 3 times, poisoning by salt fish once, cholera morbus 3 times, acute fevers 4 times, pericarditis twice, cerebral convulsions twice, rheumatism once, sunstroke once. No cause could be discovered in 35 cases. In one case the pulse was retarded to 3 per minute for several hours, in another to 4 per minute. In one of the recorded cases the pulse beat only after a lapse of thirty-five seconds and in another after twenty-five seconds.

Symptoms.—Bradycardia betrays itself at once in the examination of the pulse, which is found to beat as low at least as 40 strokes in the minute. As a rule, the slow pulse is full and hard, though it may be

feeble or scarcely perceptible. As the bradycardia is only a symptom, and for the most part of some affection of the brain or cord, the lesion reveals itself by other signs. Sometimes there is nausea with retching or vomiting, sometimes vertigo or palpitation with dyspnœa. Affection of the heart itself, especially calcification or other occlusion of the coronary arteries, is indicated by attacks of excruciating pain with extreme anxiety. Threatening arrest of the heart's action may be attended by convulsions, cyanosis, or syncope. The bradycardia which belongs to the hysterical attack, especially to the sleep attacks, is usually associated with marked vaso-motor disturbance. The patient is found in a semi-comatose condition, with pallid or flushed face and cool or cold extremities. Such a case was recently received into the hospital in the service of the writer under the mistaken diagnosis of typhoid fever.

The bradycardia of the epileptic patient occurs, as a rule, after the convulsion in the post-epileptic state. Sometimes the pulse is retarded as a premonition, and the convulsion occurs when the bradycardia is most extreme. Both the conditions, the convulsions and the slow pulse, arise from a common cause.

Paroxysmal bradycardia, which is much more infrequent than paroxysmal tachycardia, begins in the midst of apparent health with a more or less sudden retardation of the pulse, and usually in connection with nervous symptoms, emotional disturbance, palpitation, oppression. The attack subsides as suddenly as it set in, and the patient is left with a feeling of languor and depression which often ends in sleep. Other symptoms—insomnia, exhaustion, aphonia, diarrhœa, fever, paræsthesia, and paralysis—depend upon associate or causative conditions.

Diagnosis.—Examination of the pulse establishes the diagnosis and turns attention at once to the cause of the condition. Permanent bradycardia is regarded as a symptom of organic affection of the nervous system. Temporary bradycardia occurs in the course of nicotine-poisoning, lead-poisoning, icterus, uræmia, and in various infections, especially in puerperal fever. Bradycardia of reflex origin may be traced to its source, usually in some affection of the intestinal tract or of the abdominal viscera. The bradycardia which belongs to neurasthenia and hysteria is recognized by the signs of these conditions, emotional disturbance, clavus, hemianæsthesia, etc. Neurasthenia reveals itself especially in the general weakness of the whole body, in the various anxieties, phobias, and incapacities.

Prognosis is always serious. Bradycardia depends chiefly upon organic affection of the brain and cord. But a bradycardia of nervous or toxic origin, as in hysteria and nicotine-poisoning, is not necessarily so grave. The bradycardia of convalescence subsides naturally with restoration of health. The immediate outlook is determined by the condition of the heart muscle, which if strong sufficiently feeds the brain and body, notwithstanding the diminution in the number of beats. Signs of insufficiency, vertigo from anæmia of the brain, cyanosis, and dyspnœa make the outlook very grave. Complication with organic disease of the heart or œdema of the lungs intensely aggravates the prognosis.

Treatment.—The treatment should address itself to the cause.

Where this condition is irremediable, as in arterio-sclerosis, the treatment must be wholly palliative, and will consist essentially in regulating or adjusting the habits of life. The bradycardia which results from aneurysm may call for surgical intervention. Bradycardia of reflex origin may be relieved by cure of the disease of a distant organ. Thus it may be necessary to regulate the stomach, to relieve constipation, or to secure the discharge of intestinal parasites. A sluggish circulation in the abdominal organs may be quickened to activity by faradization and massage of the abdominal walls. Toxic bradycardia demands the surrender of the cause, such as alcohol, tobacco, coffee, or tea—demands also the treatment of Bright's disease, uræmia, especially by hot baths, the relief of icterus, by the extraction of gall-stones, by Krull's method of injecting cold water, the disinfection or extirpation of centres of sepsis, etc.

In the treatment of bradycardia the temptation is strong to administer the heart stimulants, especially digitalis and nitro-glycerin. But these remedies are not indicated so long as the heart supplies the wants of the body. They may be given, however, in small dose in the presence of heart failure, as indicated by cyanosis or dyspnœa. Actual collapse calls for the use of the more diffusible stimulants, caffeine, camphor, and ether; a cup of black coffee fortified with a little brandy is a strong stimulant to the heart. Clinicians find the best stimulant in camphor dissolved in the oil of sweet almonds, 1:10, of which a syringeful or two may be injected under the skin.

The more purely nervous attacks of paroxysmal bradycardia may be sometimes relieved at once by the treatment of flatulency, as by turpentine, the tincture of valerian, the milk of asafœtida, Hoffman's anodyne, etc. Milder attacks of pain may be relieved by the use of some of the modern analgesics, especially by apolysin and lactophenin—remedies which do not weaken the action of the heart—in the dose of gr. v to x. As a last resort appeal may be had to morphine, which must be used, however, with great caution in the presence of organic disease of the brain or of failure in the action of the heart. The morphine may be given alone or in combination with atropine, which would seem to be indicated as an antidote to the depressing heart poisons. Atropine, gr. j–$\bar{5}$j, is best given in solution in dose of 3 to 5 drops. Strychnine is the best general tonic to the heart, as it, by heightening the reflexes, increases the sensitiveness of the nerve centres, so that a more trivial impression is made to contribute to the sustentation of the heart.

Angina Pectoris.

Definition.—Angina pectoris (*angere*, to bind, to choke) is a disease caused by sclerosis of the coronary arteries or atheroma of the aorta at the origin of these arteries, distinguished by excruciating pain in the region of the heart, which irradiates to the shoulder and left arm, and is attended with a sense of extreme anxiety.

Etiology.—The credit of having first separated angina belongs to Heberden (1768), though the condition was recognized by Jenner in the case of the celebrated John Hunter, who finally died of the disease. Heberden ascribed the symptoms to spasm of the heart, a view which

has met with continued advocacy up to the present time. Brunton compares the condition with the spasm of a distended stomach or bladder behind an obstacle at the orifice. Eichwald attributed the pain to overstrain of the muscle in the attempt to overcome the obstacle. The character of the pain resembles that of neuralgia, and angina has been considered a neuralgia by various authorities. Piorry and Cahn located the affection in the nerves of the brachial plexus, and in our own day v. Basch subscribes to this view, explaining the anxiety as a symptom of visceral neuralgia. Peter and Lancereaux located the affection in the cardiac plexus. Romberg spoke of angina as a neuralgia of the heart. Angina has been connected also with various neuroses; thus, by Charcot and LeClerc with hysteria, and by Trousseau, who looked upon the explosion as an aura, with epilepsy. Finally, angina has not escaped implication with rheumatism. Elsner, Schmit, and Darwin looked upon the disease as a manifestation of rheumatism or of gout.

The most superficial study of the subject soon makes the fact plain that various affections have been considered under the term angina, and that at least two varieties may be distinguished: the true angina, which depends upon organic change, and the false angina, which is a mere neurosis or neuralgia of the cardiac plexus. The conditions may coexist, and the fact that explosions of angina are sometimes evoked by the cause which operates to produce a neurosis has given rise to much confusion as to the true nature of the disease. The fact is, the conditions should be considered apart, and true angina should be studied under the head of arterio-sclerosis, atheroma of the aorta, or calcification of the coronary arteries, while the false angina belongs with the neuroses of the heart.

Occlusion of the coronary arteries is quickly attended with interference with the circulation, but the effect of occlusion in producing pain cannot, of course, be directly estimated in the lower animals. It was observed by Panum that the injection of tallow and wax into the innominate artery was followed by arrest of motion of the left auricle in the course of five minutes, in the left ventricle in three-quarters of an hour, and in the right auricle in an hour and a half. Von Bezold found that the action of the heart was retarded in the course of ten to twenty seconds, became irregular in the course of forty-five to one hundred and fifty seconds, and hereupon in a short time ceased to beat entirely. Samuelson saw that the right side continued to beat longer than the left, and that in this way the left auricle was filled to distention in such extreme degree that it might be regarded as the cause of the pain of angina. Cohnheim and Schulthess-Rechberg observed no immediate influence upon the motion of the heart after ligating a large branch of the coronary artery, though arrhythmia and retardation set in by the end of the first minute. During the second minute both ventricles ceased to beat at once. Release of the ligation after arrhythmia had set in failed to restore the action of the heart, which had become completely paralyzed. This paralysis could not therefore be attributed to mere anæmia or to defective oxygenation or increase of carbonic acid gas, but could be explained only as the direct effect of some poison which under a perfect circulation is carried off and eliminated.

PLATE I

Angina Pectoris. Sclerosis at Orifice of Left Coronary Artery. General Atheroma of the Aorta. The Glass Rod *C* is introduced into the open Right Coronary Artery. The Obliterated Left Coronary *C'* barely permits the penetration of a Bristle. (Huchard.)

Charcot made a special study of incomplete and irregular occlusion of the arteries in the extremities in the process which he distinguished as *claudisation intermittente par oblitération artérielle*. In experiments upon the common iliac artery in this way it would seem that occlusion produces no distinct effect so long as the patient remains perfectly quiet, but that pains set in with exercise, along with a feeling of coldness, numbness, spasmodic contraction, and weakness. So partial occlusion of the coronary arteries may be unattended with any disturbance on the part of the heart until some extra demand is made for increased action. In this way any physical strain or psychical excitement may precipitate an attack of angina pectoris.

But sometimes typical attacks of angina have been known to occur in individuals who showed upon post-mortem examination no lesion of the coronary arteries. In a certain number of these cases, however, atheromatous changes have been found in the aorta and in the direct vicinity of the orifices of the coronary arteries. In fact, atheromatous degeneration of the aorta is a frequent lesion in this condition. The fact that the aortic valves alone are frequently found affected in angina pectoris speaks decidedly for the localization of the degenerative process. Potain found patches of aortitis localized exactly at the orifice of the coronary arteries, and Huchard depicts cases of occlusion of the orifices of the coronaries by plaques of atheromatous matter. (See Plate I.)

But sclerosis of the coronary arteries does not necessarily produce the symptoms of angina pectoris. Sometimes the sclerosis does not block the vessel to sufficient extent to greatly interfere with the circulation; in other cases the circulation is carried on by collateral or supplementary coronaries. Tapret and Bador recorded cases of this kind. So that sclerosis of the coronary arteries is not necessarily incompatible with normal action of the heart. V. Basch declares that severe cases of angina justify an absolute diagnosis of sclerosis of the coronary arteries. The cases of angina independent of all organic lesion fall under the head of pseudo-anginas.

Predisposing Causes.—*Age and Sex.*—As the true angina depends upon the process of sclerosis, the cause of the disease will be found in the conditions which produce or favor the development of degenerative change. Arterio-sclerosis belongs among the changes of age. It is not surprising to learn, therefore, that the majority of cases occur in advanced life. In fact, Quain found that 80 per cent. of the cases developed after the fortieth year of life. Balfour found the evidence of sclerosis in one sixth of all the senile hearts which he examined. But the changes of senility are often precipitated in earlier life, especially by alcohol, syphilis, and gout, so that well marked angina may occur even during adolescence. These are causes also which operate chiefly in the male sex. Only 8 of the 88 cases cited by Forbes and only 7 of the 74 cases quoted by Lartigne were women. In the statistics collected by Balfour five sixths of the cases occurred amongst males, and Lusanne puts the proportion as high as 97 per cent. in men. This remarkable disproportion illustrates the fact that other factors, especially the influence of alcohol and syphilis, are of more importance in producing the changes of arterio-sclerosis than age.

Heredity.—There is no doubt that certain individuals inherit a tend-

ency to premature sclerosis, so that heredity is not without influence in the production of angina pectoris. Most of these cases, however, are transmissions of the neurotic temperament, and fall more directly under the head of pseudo-anginas.

Syphilis.—The influence of syphilis is unmistakable. Syphilis exercises a direct toxic influence upon the walls of the bloodvessels. As stated elsewhere (page 534), it produces such degeneration as leads to aneurysm; in other cases the degeneration is more diffuse and results in atheroma. An indirect influence is exerted by syphilis in the cases of angina which occur in the course of tabes dorsalis. Leyden finds an explanation of the angina in these cases in the direct implication of the pneumogastric nerve. Charcot had called attention to these attacks of angina under the cardiac crises. These crises may alternate with the gastric crises, which are also ascribed to organic changes in the pneumogastric nerve.

Habits.—As bad habits or the diseases which they engender are very liable to occur in this condition, it is easy to understand that certain cases represent mixed forms. Leflaire maintains that angina is more frequent in clerical life—among preachers, physicians, writers, etc.

Pseudo-angina occurs more especially in connection with hysteria and neurasthenia—conditions in which heredity and bad psychical surroundings play the most important part.

The pseudo-angina pectoris, which is a more distinct neurosis, may be attributed to direct, reflex, or toxic cause, but two or all of these causes may be combined in an individual case.

Direct Causes.—Under the direct causes fall the cases which are produced by affection of the cardiac branches of the pneumogastric nerve, as well as the cases which have cerebral origin from the disturbances in the cortex cells which give rise to the symptoms of hysteria and neurasthenia. Both these disturbances may arise from physical or psychical strain, especially from emotional excitement, or may be evoked in turn by reflex or toxic cause. Mackenzie shows by several illustrations that the pain and hyperæsthesia in the wall of the chest and in the left arm which occur in the course of many heart diseases show a remarkable resemblance in distribution to herpes zoster, and, as shingles may be referred to a disturbance (from infection) in the posterior roots, especially to the spinal ganglia, Mackenzie is inclined to locate the cause of the attacks of pain in heart disease in the same region.

Causes of reflex origin are most common in the course of the alimentary canal, and are associated especially with nervous dyspepsias. The connection is recognized in the so-called angina dyspeptica. Thus attacks of pseudo-angina may occur as the result of an acute indigestion or more frequently from gaseous distention with mechanical interference with the circulation. Fruitful causes are found also in affections of the uterus and ovaries, and attacks of angina are not uncommon in the course of dysmenorrhœa, salpingitis, etc.

The *toxic causes* play a more frequent and important rôle. Among these causes are to be considered the action of alcohol, tobacco, tea, and coffee. *Alcohol* furnishes an illustration of conjoint cause. Thus the ingestion of large quantities of the lighter forms leads to plethora. Beer-

drinkers especially suffer with derangement in the action of the heart, sometimes expressed in pain. Alcohol is a frequent cause of arterio-sclerosis, aortitis, and atheromatous degeneration of the coronary arteries. Alcohol also directly poisons the nervous system and irritates the terminal filaments of the nerve fibres of the heart. Alcohol first stimulates and then exhausts the heart.

Tobacco has an even more distinct action upon the muscle and nerves of the heart. Even in small dose nicotine retards the action of the heart and renders it weak. Chronic nicotism affects also the nerve centres. It produces a form of neurasthenia marked especially by insomnia, impairment of memory, and vertigo, and shows its influence on the heart, especially in palpitation, arrhythmia, and angina. The influence of tobacco has long been recognized as the most common toxic cause of angina, and this influence is most marked in cases of habitual smokers, especially of strong cigars, and in individuals who masticate cigars continually. The habit of repeatedly relighting cigars and of smoking cold stumps of cigars saturated with saliva finally induces nicotism in the most refractory subjects. The most successful cases in therapy are those in which attacks so severe as to simulate a real angina are brought under control by the simple abandonment of all use of tobacco.

Coffee and tea exhaust the heart in the same way, but in much less degree. Caffeine and theine are both stimulants to the heart, but in excessive use exhaust it. Attacks of pseudo-angina are thus not infrequently observed among individuals who lead clerical lives, especially when the nervous system is forced to extra work at night by the stimulation of strong coffee. Angina ranks among the prominent symptoms in aggravated cases of the so-called tea-drinker's dyspepsia.

Among the toxic causes must be cited also the influence of *gout* and *lead-poisoning*, conditions which have been long recognized under the head of *angina uratica* and *angina saturnica*. The poisoning in both cases exerts its influence directly upon the heart nerves, and in both of these affections real angina may develop through the intervention of arterio-sclerosis, which occurs so commonly in both gout and saturnism.

Symptoms.—The attack of true angina pectoris occurs suddenly. The patient without premonition is literally stabbed and transfixed with pain in the region of the heart. But the cause of the disease is of slow development. The fact is that, while the attack is of sudden occurrence, there will have been evidence of failing circulation or interference with the action of the heart for some time, usually for a long period, previously. True angina pectoris does not set in suddenly in the midst of perfect health. It depends, as stated, upon the sclerotic process which directly or indirectly involves the coronary arteries, and this process of exceedingly insidious development finally makes itself manifest in some disturbance of the heart. For the most part, the patient will have felt for a long time some interference with the circulation. He suffers from palpitation or arrhythmia. There is a feeling of weak action, of intermission, of interruption. The patient becomes short-winded—a fact which is observed first by the suburban resident in running to catch cars or trains, or is observed in the first attempts with the bicycle. During the process of adjustment after this effort there are

palpitation, uneasiness, and distress. The heart takes a longer time to adjust itself; moreover, disturbance in the action of the heart is excited by apparently trivial cause, as by emotional excitement, physical effort.

The paroxysm or explosion of angina occurs during the action of some such cause, as in climbing a hill, in breasting a wind, in arising to make a speech in public, during some emotional excitement—as a fit of anger—in coitus, in straining at stool, etc.

On the other hand, certain cases set in more spontaneously, as during perfect rest, at table, or even during sleep.

Death is sometimes sudden in these cases—so sudden, in fact, that there may be no time for the appreciation of pain or other symptoms. Thus, patients have been discovered to be dead in the night, or have been found dead in the morning by bedfellows who have not been awakened from sleep. In one case in the experience of the writer a patient fell dead in the act of unrolling a bandage applied for the relief of varicose veins in the leg. In these cases the heart is paralyzed, and contraction ceases exactly as after artificial occlusion of a main branch of the coronary artery.

In other cases the patient feels the pain, seizes the region of the heart, becomes suddenly cyanotic, and falls into syncope, which may terminate fatally in a few minutes. For the most part, however, the action of the heart is not so completely interrupted. The pain in these cases is agonizing in the extreme, and is usually associated with a feeling of constriction or pressure. Sometimes the patient speaks of a direct pressure, as of the insertion of a wedge or of the bending backward of the sternum. Balfour described the sensation "as if a mailed hand grasped the chest in the cardiac region and squirted through its fingers flashes of excruciating agony up to the left shoulder-joint." Thus the pain radiates to the shoulder and down the left arm, sometimes only as far as the elbow, or again along the course of the ulnar nerve to the third and fourth fingers of the left hand. More infrequently the pain takes another course, toward the right shoulder and down the right arm, or backward toward the spine or scapula or to the region of the occiput. In rarer cases the pain shoots downward in the abdomen or to the lower extremities. The irradiation takes place along the line of the nerve trunks, and the localization in the shoulder and arm is due to the inosculation of the cardiac plexus with the anterior branches of the fourth upper cervical and first thoracic nerves, and of the first thoracic nerves with the lower fibres of the brachial plexus. The preference of the left side may be ascribed to the greater frequency of affection of the left half of the heart. All motion of the body is at once arrested, and the excruciating agony that is felt reveals itself in the expression of the face.

Associated with this pain is the extreme anxiety which with the sensation of constriction in the chest has given the name to the disease. The anxiety is a cerebral sign. It is the appreciation on the part of the brain of the imminent danger of arrest of the action of the heart. Such an anxiety is sometimes seen in delirium tremens, melancholia attonita, typhoid fever, endocarditis, and other affections entirely independent of pain. It may be ascribed in all cases to the action of toxins upon the nerve centres.

The action of the heart itself during the attack is very variable. Sometimes the circulation is entirely unaffected; sometimes there is palpitation or arrhythmia. The pulse may be increased or decreased in frequency; it is often entirely unaffected. Brunton always observes with the sphygmograph increased tension in the arteries, but, as Eichwald noted of an increase in the pseudo-anginas of hysteria, there is no necessary connection between the rise of blood pressure and the true angina. At the same time the evidence of disease of the heart may be discovered in the majority of cases. Thus, Walshe found the evidence of some heart lesion in every one of his cases.

The most frequent valve lesion is some affection, especially stenosis, of the aortic valves, dependent upon atheromatous change at the base of the aorta. But the evidence of chronic myocarditis may be made out in most cases.

Respiration may remain perfectly normal during the attack. It is generally more frequent and shallow in the unconscious inhibition of all effort on the part of the patient. Anything like real dyspnœa may occur only in connection with some organic affection of the heart.

The posture is peculiar. In the presence of extreme suffering most patients seek instinctively the recumbent posture. This is not the case, however, in angina pectoris. The patient sits up or stands up, because the upright posture distinctly diminishes the arterial tension.

The duration of the attack varies from a half minute to a half hour or even to several hours. In a case reported by Huchard the attack lasted as long as thirty-six hours. Remissions occur in cases of such long duration to present the appearance of rapidly successive attacks or so-called subintrant forms.

The interval between the attacks varies greatly. In exceptional cases the patient has but one attack, from which he recovers entirely. It may be assumed in these cases that the occlusion, as by an embolus, has been relieved or that collateral circulation has been established. As a rule, however, the attacks recur. In the case of John Hunter there was an interval of three years between the first and second attack. In a later period of life the attacks became more frequent and were followed at times by syncope. In this case the angina was precipitated by any over-exertion or emotional excitement. The last attack, which was followed by almost sudden death, occurred after an excitement of this kind. In other cases the patient may be able to walk a few steps, or, as in an instance reported by Fräntzel, even for some distance.

The attack subsides suddenly, as a rule, leaving a feeling of prostration, with sensations of numbness and formication in the arm.

These various symptoms are characteristic of the pseudo-anginas, but it will be remembered that the real lesion and the neuroses are sometimes combined, so that vaso-motor changes are encountered also in cases of real sclerosis.

Pseudo-angina.—Pseudo-angina is distinguished by its occurrence in connection with hysteria and neurasthenia. There is in this affection also predominance of symptoms on the part of the vaso-motor system. There is pallor of the surface, sometimes of the arms and fingers. Heberden describes asphyctic conditions of the left forearm and hand. This state is marked by coldness and insensibility of the surface, with

the sensation of swelling. Trousseau saw discolorations of the surface. The arm and hand assumed a purplish tint after a state of pronounced pallor. V. Basch reported the case of a patient who described the sensation as if cold water were poured over the lower extremities, and Fränkel saw cases in which the sensation of numbness and deadness was associated with feelings of formication.

Where such changes assume prominence the condition is distinguished as a vaso-motor angina. Nothnagel found cases of peculiar anæsthesia in which the surface was insensitive to puncture by a needle or to thermic impressions; these same cases are, however, usually peculiarly sensitive to cold, and the mere washing of the hands and feet in cold water may suffice to develop an attack. So a seizure may set in on first entering a cold bed.

Pseudo-angina ceases after the manner of hysterical explosions, often with eructations of gas or with the discharge of large quantities of urine—*urina spastica*.

Diagnosis, as a rule, is not difficult. The excruciating pain, with the peculiar anxiety which sets in suddenly in a man past the meridian of life, who shows and has shown usually the signs of arterio-sclerosis, sufficiently distinguishes the disease. Pseudo-angina occurs most often in the female sex, and is evoked by emotional excitement, the evidence of which is shown during the attack. Patients affected with pseudo-angina may walk up and down the room wringing the hands, throw themselves upon the bed, etc. Real angina is distinguished by the fact that the suffering is too great for such display of emotion. The patient sits or stands perfectly quiet, and actual agony is unmistakably depicted in the face. The radiation of pain in pseudo-angina is more irregular, as it is by no means confined to the region of the heart and to the left arm, but may extend over the whole side of the body. The presence of other signs of hysteria, hemianæsthesia, aphonia, globus, clavus, hysterogenic zones, etc., aid in the diagnosis.

Pseudo-angina occurs also in neurasthenia, which belongs to a younger period of life and is marked by a general weakness, with peculiar anxieties, agoraphobia, phosphaturia, etc.

The neuralgic affections which are produced under the toxic effects of alcohol and tobacco show themselves also at an early period of life, with an odor or other evidence of the abuse of alcohol and tobacco. Lead-poisoning and gout are frequent causes of real angina, but obstinate neuralgic affections of the heart may occur in connection with either disease, independent of coronary sclerosis. The lead line on the gums, the obstinate constipation, colic, arthropathy, and paralysis distinguish poisoning by lead, while the affection of the joints characterizes the attack of gout.

In a general way it may be said that the pain is never so severe in a pseudo-angina as a real angina. In fact, pseudo-angina is characterized rather by the predominance of paræsthesia over pain and by the presence of vaso-motor phenomena.

Cardiac asthma is distinguished by attacks of dyspnœa, which set in especially after effort, is attended with cyanosis of the face, enlargement of the liver, and dropsy. Cardiac asthma is generally due to œdema of the lungs, which in turn is caused by failure on the part of the left

ventricle. Both these conditions reveal themselves by distinctive signs. Œdema of the lungs is marked by the universal presence of moist râles, especially in the postero-inferior region of the chest, and heart failure may be distinguished in the pulse, which is usually not much altered in a case of real angina.

Intercostal neuralgia is sometimes mistaken for angina, but only under superficial observation. The pain of intercostal neuralgia is unattended with anxiety, and is in the worst cases by no means so severe as the lightest case of real angina pectoris. Intercostal neuralgia may be further distinguished by the painful points at the spine, in the axillary line, and near the sternum, and by the quick relief which is usually afforded under galvanization of the chest.

Prognosis is different in the two affections. On account of the nature of the lesion it is always grave in real angina. An artery whose walls have suffered sclerosis is liable to rupture. Gallardi reported a case in which death occurred from rupture of the right coronary artery. The celebrated sculptor Thorwaldsen died in the theatre in this way. Again, the gradual increase of the process leads to eventual occlusion by thrombus; moreover, the arterio-sclerotic change may be usually found elsewhere, and often in the cerebral arteries or in the arteries of the kidneys. Lewis and Gore reported cases associated with calcification of the cerebral arteries. As already stated, the first paroxysm may be fatal or death may occur in any subsequent attack. Hollister reported a case in which the first attack was fatal in twenty hours.

At the same time, it must be appreciated that the interval between the paroxysms may be increased and the severity of the attack mitigated under appropriate regimen and treatment. Finally, a collateral circulation may be established in the heart, so that the patient may eventually entirely recover. Flint reports a case of this kind, and Curschmann two cases in which a subsequent retardation of pulse was left as the sole relic of former angina. Bendel reported a case associated with loss of speech, which ended in recovery. But how grave a case may be and nevertheless recover is illustrated in the report by Labalbary of one which occurred in connection with a general arthrodynia, and which was attended by grave icterus, epistaxis, and purpura, but which was followed by complete recovery.

Pseudo-angina has a favorable prognosis in so far as life is concerned. Cases dependent upon reflex or toxic cause may recover entirely with the removal of the irritation or the abandonment of bad habits. For the most part, the disease is obstinate with the obstinacy of hysteria and neurasthenia, though most patients recover from the symptoms of the pseudo-angina.

Treatment.—The treatment must be addressed to the attack and to the intervals, and, as regards the interval at least, is different in the two forms of the disease. During the attack every effort is made to reduce the tension in the vascular system, and thus to relieve the heart of extra work. Absolute rest is observed of itself. The patient instinctively takes the standing or sitting posture, as the blood pressure is increased in the recumbent posture.

The remedy par excellence to secure this result is amyl nitrite, which was introduced into therapy by Guthrie in 1859. This drug dilates

the peripheral vessels and releases the tension in the heart. The action of the remedy becomes visible in the flushing of the face and fulness of the head, which is associated at times with a light degree of vertigo and intoxication. At the same time, the heart begins to throb and pulsation is felt in the arteries. The attack of angina now ceases at once, and the patient feels immediately relieved of the excruciating suffering. Further use of the remedy should be discontinued, as longer persistence increases the sense of fulness in the head up to headache and stupor. The remedy is best given by inhalation in the dose of 2 to 5 drops, which may be dropped from a bottle or, more conveniently, released by crushing between the fingers very thin glass tubes, so called "pearls," in which it is contained hermetically sealed. Where the attacks recur the use of the drug may be repeated. Any protracted or recurrent attack may necessitate the employment of larger doses or more continuous inhalation. Balfour described the case of a patient who suffered with angina in connection with aortic regurgitation, and who got relief by inhaling the remedy during the day, but, not content with this quantity, he "used to soak his pocket handkerchief with amyl nitrite and go to sleep with it lying on his face, without any ill results." Certain individuals seem to be thus insusceptible to the toxic effects. Amyl nitrite controls most of the cases of real angina, but it sometimes fails. So thoroughly acknowledged is the value of the remedy that Fagge reported as something unusual a case of angina pectoris in which the amyl nitrite was used without benefit. Harder cured a case of pronounced angina with the nitrate of silver.

The anæsthetics proper, and especially ether and chloroform, have long been used in the treatment of angina. As there is danger of heart failure, ether, on account of its stimulating effects, is to be preferred to chloroform. Romberg preferred to pour the ether, a teaspoonful or two, into a saucer and have the patient inhale the fumes. Fränkel reported the case of a physician who controlled nocturnal attacks with which he was regularly seized in this way. On account of the danger of the anæsthetics they should never be used except in cases where the nitrites fail.

Nitro-glycerin is a remedy of great value in preventing the attacks, though, because of its slower action, it is not so efficacious as amyl nitrite in arresting an attack. Nitro-glycerin is of especial value in the attacks of repeated occurrence and in the lighter forms of the so-called vaso-motor angina or the different varieties of pseudo-angina. It acts in the same way by dilating the capillaries, but the action is longer sustained, sometimes as long as six or eight hours. Nitro-glycerin is best administered in a 1 per cent. solution, of which one or two drops may be given in a teaspoonful of water or wine. A convenient form of administration has been devised in chocolate tablets, of which each one contains $\frac{1}{200}$ gr. nitro-glycerin. Great tolerance may be gotten under continued use, so that the remedy may be pushed to an extreme degree. Murrell once gave 110 minims of the solution—that is, more than 1 minim of pure nitro-glycerin. Certain cases secure perfect exemption from attack under the continued use of this drug over a period of months.

Sodium nitrite was recommended as a substitute by Hey as having

similar effects, in that it liberates nitric acid *in statu nascendi*, upon which the virtue of all the nitrites is believed to depend.

Nitro-glycerin may be very conveniently given also in solution with the nitrous spirits of ether. Thus:

℞. Nitro-glycerin, ♏j;
Spiritus ætheris nitrosi, ℥iss.—M.
Sig. Dose, fifteen to twenty drops during the attack.

The sodium nitrite may be taken regularly in the way of prevention of attacks, as follows:

℞. Sodii nitritis, gr. viij–xv;
Aquæ, ℥v.—M.
Sig. Dose tablespoonful three times a day.

The most obstinate and protracted cases may demand the use of morphine, which should, however, be given with great caution during the attack, that the death which occurs so often in angina be not attributed to the drug.

Cases of pseudo-angina are relieved by simpler means and by the use of the various antispasmodics—valerian, the bromides, Hoffman's anodyne, etc. Sinapisms applied to the præcordium help to secure relief. The application of hot and cold water proves of great value in certain cases. Fränkel prefers ice bags or cold cloths, and Schott heat in the form of hot water applied to the region of the heart. The attack is often cut short by the immersion of the hands and feet in hot water, as hot as it can be borne, or by the application of mustard plasters until the skin is reddened, or by dry cups upon the chest and back. More severe cases call for the use of morphine, which must be given with caution, because of the danger of associate heart weakness which may have been unrecognized, and because the differential diagnosis between the two forms is not always clear.

The underlying condition must be addressed in all cases. Where there is evidence of arterio-sclerosis the iodides should be regularly administered in small dose over a long period of time. In the absence of any fatty change arsenic is indicated also in small dose. Finally, the patient must learn to control his emotions as well as his motions.

DISEASES OF THE BLOODVESSELS.

By JAMES T. WHITTAKER, M. D.

GENERAL VASCULAR DISORDERS.

Angio-neuroses, Etc.

The study of the changes which take place in the walls of the bloodvessels is the study of the ultimate processes of life, for the natural dissolution of the individual in the process of so-called physiological decay shows itself first in the vascular walls. In the contemplation of these structures we are brought to face the problem of life itself, and stand upon the confines of knowledge, beyond which it is at present impossible to penetrate. We content ourselves, therefore, with the observation of the changes which are visible to the eye. These changes are called structural. Beyond these changes are the so-called functional (molecular) changes, which lie at present largely in the realm of speculation, and may not, hence, be considered here.

The bloodvessels have much higher dignity than is commonly assigned to them. They may no longer be regarded as mere tubes for the conduction of the blood. They are eminently living structures, thoroughly differentiated, endowed with power of contraction and dilatation, highly nourished, extremely sensitive, quickly responsive to every call. Moreover, they have to do directly with the ultimate processes of oxygenation, nutrition, and metabolism. They are not to be looked upon as simple conduits for the circulation or as mere appendages to the heart. On the contrary, the heart is rather an appendix to the bloodvessels. Low in the scale of life the circulation is sustained without a heart, and in the rising scale the heart originates as a local development of the bloodvessels.

Neudörfer suggests that the dilatation as well as the contraction of the arteries may be an active process. It may be permitted, he says, to regard the orbicular muscle of the heart as so disposed that the primitive striated muscle fibres are wound about the endocardium in layer after layer like thread upon a spool, so that it makes circles and continuously longer and more oblique ellipses; while the layers which are attached to the fibres and tendinous parts of the heart make no such closed circles or ellipses, but, like other striated muscles of the body, may induce a change of position from points of attachment.

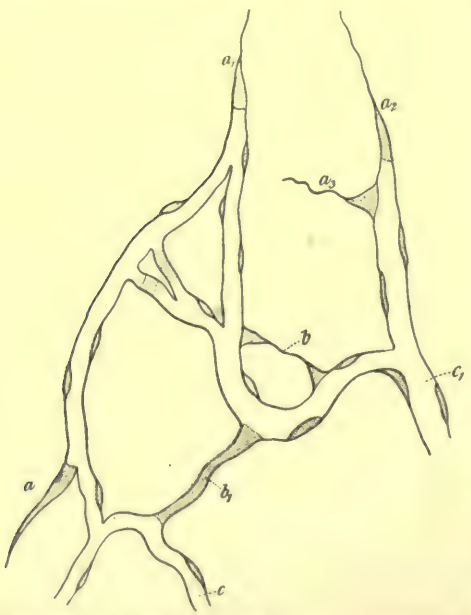
This arrangement of the muscle fibres leads to the view that the diastole as well as the systole is the result of an active contraction. The idea that the dilatation of the heart is active, and is not, as usually believed, merely passive from relaxation of muscle fibre, better corre-

sponds to the observations of the physiology and pathology of the circulation. Under this view the heart acts not only as a force, but also as a suction-pump.

The arteries as well as the heart show rhythmical contraction, so that the arteries are independent of the action of other organs. The dilatation of the arteries may be also active, so that the vascular system is a reservoir with the arrangement of a force-pump which discharges blood into the arteries, and at the same time of a suction-pump which withdraws it from the veins, arteries and veins sharing in the acts.

The Development of the Vessels.—In the beginning, as in embryonic life, the bloodvessels are formed first by an aggregation of cells in the middle germinal layer, which constitutes a kind of network in the area pellucida. Oval and round cavities, with smooth inner surfaces and clear contents, develop in these cells as the first intimation of capillaries. The contents would appear to be a secretion which stands under a certain degree of pressure. Outshoots from the wall of these capillaries constitute the secondary vessels which in the course of time traverse the body as the bloodvessels. The heart is developed later, and in its first contractions propels in the capillaries the fluid which now receives color and is called blood. Thus the development of a vascular system consists, first, in the construction of a capillary network, and afterward of arteries and veins in proper relation; and the working capacity of the heart is dependent on the quantity and pressure of the blood in the great vessels. In other words, the tissues determine the rapidity of the circulation. The lumina of the arteries and veins in the domain of the capillaries and the action of the heart are equivalents of the demand of the tissues (Thoma).

FIG. 23.



Development of the blood vessels: a, a_1, a_2, buds united at b, b_1; open at c, c_1 (Thoma).

So soon as the body of an individual reaches maturity it begins to show evidences of decline and decay, and this evidence is shown first in the vascular walls. Alterations of structure may be noticed here, in the absence of any disease, often as early as the age of thirty-five, though at this early period the process seldom assumes magnitude enough to interfere with function. But disease rapidly precipitates or intensifies these changes, and death is due to them, directly or indirectly, in the great majority of cases.

The walls of the vessels, which are usually divided into three layers,

have a very complex construction. The outer wall, chiefly fibrous, carries the nutrient vessels and gives the structure strength. Thoma has shown that Pacinian corpuscles occur in great number in the adventitia of the larger arteries, where they probably serve as sensitive organs for the perception of variations of pressure. The middle wall is muscular and elastic; it receives the nerve supply, and is the essential factor in the regulation of the circulation and the distribution of the blood. Bonnet objects to the usual division into intima, media, and adventitia, and adopts the six layers distinguished by Henle—1, endothelium; 2, long fibres; 3, fenestrated membrane s. elastica interna; 4, circular fibres s. media of the later authorities; 5, elastica externa; 6, adventitia. By staining the elastic fibres with orsein Bonnet was able to demonstrate that the elastica interna and externa are nothing else than the strongly developed lamellæ of the media. The elastica interna and externa do not belong, therefore, either to the interna or the adventitia, but both to the media. With this fact coincides the condition of the artery walls and elastic substance in the developmental period. When the muscle tissue becomes enfeebled from any cause the vessel loses its fine adjustment, and later its power of resistance to the impact of the heart; the vessel now suffers dilatation like any ordinary inert, yielding tube. The inner wall is a layer of apposed flat cells, which offer but little resistance to the circulation, and are endowed with a peculiar kind of osmosis—really a secretion—which in the smallest vessels or capillaries ministers to metabolism and forms part of the phenomena of life.

Up to the year 1866 it was believed that the capillaries were of constant size. In this year Stricker showed that the capillaries are contractile, that their lumen becomes narrower under electrical irritation—that the resistance offered by the vascular system is therefore not constant, but changeable, and may raise or lower the blood pressure.

The influence of the nervous system is shown in the changes in the blood pressure. Bezold found that when he cut through the spinal cord and excited the peripheric end the blood pressure rose. He ascribed this increase to irritation of the heart nerves which arise in the cervical cord. Ludwig and Thyri showed, however, that the same increase of pressure occurred when they cut through all the nerves of the heart. They found the cause of the increase in the contraction of the vessels of the abdominal viscera and of the skin of the lower extremities. M. and E. Cyon demonstrated later that no increase of pressure occurred when the splanchnic was cut through. Hence the increase of blood pressure must result from the fact that the vessels of the abdominal viscera contract and empty themselves of blood. With the discovery of the depressor nerve, centripetal irritation of which sinks the blood pressure in high degree, Ludwig and Cyon found at the same time the cause of its effect in dilatation of the vessels of the abdominal viscera and increase in the quantity of blood in these vessels (Federn).

What, cries Benedikt, is the particular driving power of the circulation? Any one will answer, he says, The action of the heart as excited under the influence of the nervous system. But whence is the power of the nerves? The answer reads, The driving power of the circulation depends upon the biochemical hunger and biochemical aversion of the

tissues. The attractive and repelling action of the cells is governed by the nerves and end organs, and is conducted to the muscle of the heart. Then the question arises, Is the neuro-muscular driving power limited to the heart? We know this not to be the case, as we observe local disturbance in the circulation, and, as Ludwig said, "The organism may bleed into any large organ." There must be, therefore, local hearts, and this is found in the muscular tissue of the vessels. Now the question arises, Whence is the driving power for these local hearts? and the answer is, In the cells, which must express their wants in particular nerve tracts. These local hearts have, moreover, immediate connection with the central heart. V. Basch found that the pressure in uniform arteries was not always the same.

In the case of insufficiency of the aorta, where the tissues receive too little blood, local hearts take up the circulation, so that the local vessels undergo extreme dilatation. This process may be considered as an increased aspiration on the part of the individual organs.

By means of the onychograph, a kind of sphygmograph attached to the nail of the finger, Herz found it possible to study the condition of the finest vessels. In nervous individuals there is a rapid simultaneous change in the diameter of the smallest vessels entirely independent of respiration. External influences make themselves felt. Cold changes the onychogram into a straight line; heat, especially radiating heat, excites great pulsations.

Concentrated tension makes the pulse smaller and more indistinct in its details. Abdominal massage and hydriatic methods show their influence very markedly upon the smallest vessels.

Pathological conditions have varied effect. The visible capillary pulse in icterus, known as the symptom of Drasche, makes itself manifest in the onychogram by a maximal pulse. This dilatation, which is evident in the finest vessels, would seem to refer to a paralysis.

Fever alters the vessels. In chills (malaria) the sphygmograph shows, in consequence of the increased respiration, a coarse wave line without pulsatory variations, corresponding to a spasm of the vessels. After it appears a great pulse which shows relaxation of the vessels.

Of diseases of the vessels, arterio-sclerosis was especially studied. The results obtained do not support the view that this disease originated from the periphery of the vascular system, for in cases where the aorta only was sclerotic a large radial pulse was found in connection with extensive pulsatory variations at the finger-nail. But in advanced cases of arterio-sclerosis the smallest vessels become rigid, and the nail pulse almost entirely disappears.

The influence of the nervous system upon the walls of the vessels, aside from physiological actions, is shown in the alterations considered under the vaso-motor (including trophic) disturbances. We may not stop here to consider whether or not there is a special centre for, or whether or not there are special, vaso-dilator nerves. The tendency at present is to explain both the contraction and dilatation by the action of the vaso-motors under excitement or paralysis. The vaso-motor centre has been located in the medulla, and quite exactly, in the rabbit, in the upper olivary body. The nerves issue through the lateral columns and emerge chiefly by the anterior roots, collecting into the

sympathetic. According to Goltz, different parts of the body have special reflex vaso-motor centres in the spinal cord. That the cerebrum itself has to do with the size of the vessels is proven by the phenomena of blushing and pallor and by the anomalies of the circulation in hysteria. Persistent redness of the skin, with heat, swelling, and pain confined to a single extremity, constitutes a distinct disease, described by Weir Mitchell as erythromelalgia. Persistent spasm of the extremities may constitute also the condition known as spontaneous symmetrical gangrene (Raynaud's disease), though this condition, with that of senile gangrene, is better accounted for by the changes which occur in arterio-sclerosis.

Leubuscher found that in cases of diffused brain disease in children vaso-motor disturbances of the most varied kind were shown in the majority. Most of these patients complain of cold hands and feet. The skin is cold in the peripheric parts and shows a cyanotic appearance. The fingers and toes look as if frozen, and that, too, in the warmest periods of the year. In some of the cases of acute dementia œdema had developed. The superficial arteries may be felt narrow and strongly contracted through the skin. Many cases show indistinct flushing due to an abnormal excitability of the peripheric vessel musculature—that is, of the vessel nerves. The vaso-motor disturbances run parallel with the psychical condition. During psychical activity the irritability of the vaso-motor apparatus is increased, the contraction of the small arteries more intense, irregularity in the action of the heart more distinct. The more or less permanent spasm of the small arteries, which is itself dependent upon brain disease, produces a permanent interference with the circulation, which leads to an increased activity of the heart, with final hypertrophy.

Bright's Disease.—The cause of hypertrophy of the heart in Bright's disease is now ascribed to irritation of the vaso-constrictors by the excrementitious substances accumulating periodically in the blood, which fail to be eliminated on account of the defective excretion of the kidneys. The retention of these excrementitious matters is shown in an increased contraction of the vessel wall demonstrable by the sphygmograph, and in the greater and greater increase of the blood pressure, the natural consequence of which is hypertrophy of the left ventricle and thickening of the vessel wall, of the muscularis, and of the intima. This hypertrophy of the heart is an important compensation in beginning reduction of excretion of urine, as the amount of excretion of water in the kidneys depends upon the rapidity of the circulation in the glomeruli and indirectly upon the blood pressure in the renal arteries. The increase of the water of the urine is attended not only with an increase, but also probably with an increased imbibition by the epithelium, of the convoluted tubules of the urea (of urea including masses of the organic constituents of the urine); further, the casts accumulated in the urinary tubules are thus easily washed away (Leube).

Acute angio-neurotic œdema—a condition in which acute swelling occurs suddenly, especially about the eyelids or face or the hands—is another evidence of vaso-motor influence. Such an œdema in the larynx may even cause suffocation. There is good ground for believing also that myxœdema, while a direct expression of disease or destruction of

the thyroid gland, as a cachexia strumipriva, characterized by a peculiar mucoid swelling of the skin with special trophic and sensory disturbances, has its ultimate origin, like Basedow's disease, with which it has points in common, in the spinal cord.

Neurasthenia.—Runge remarked upon the frequency of changes in the temporal arteries in neurasthenic patients. This author also observed ectasia of these arteries in one third of these cases. The dilatation was usually unilateral. Löwenfeld states that the condition really belongs to the changes of arterio-sclerosis, which is often precipitated by alcohol, gout, and nephritis.

The investigations of Anjel with the plethysmograph showed graphically the changes in the central motor apparatus and in the peripheric vessels. Anjel found, as Mosso had observed before, that every emotional excitement and sensitive irritation produced, on the one hand, diminution in the volume of the arm and an increase in the volume of the brain—conditions which continued as long as the irritation or excitement lasted. Beard found in the majority of cases of neurasthenia alteration of the arterial tension, marked especially by distinct and persistent reduction. Weber went so far as to distinguish three groups of cases of varying degrees of tension. Bouveret long ago noticed transitory œdema in the hands and feet of neurasthenic women, and Rambielinski remarked upon the flying œdema of this condition. The swelling occurred in one case on the hand, in another on the face, assumed a certain density, and then disappeared in a quarter of an hour, to leave no trace, but to repeat or renew itself after emotional excitement.

Hysteria.—Intense flushing or rapid change from pallor to flushing of the face under trivial or emotional excitement is a very common appearance in hysteria. This change occurs frequently also as an aura symptom of hysterical attacks. Quickly changing pronounced redness or rapidly recurring redness of the face or ears during lethargic attacks announces the occurrence of more violent convulsions. In the course of hysterical arthralgia Brodie observed several times in the course of the day recurring variations in the vaso-motor innervation of the extremities—at first, coldness and pallor for several hours, then redness, increased temperature, and sweating. In the case of a woman aged twenty-three Rosenthal saw as a premonition of an hysterical attack discoloration of the hands with subjective feeling of coldness. The temperature, which on the days free of attack was 94° F., sank to 87° F. during the attacks, while the hands became white, the ends of the fingers and nails deeply blue. After an attack the temperature arose to 96–96.6° F. The fingers and nails became very red, and an active transpiration showed itself in the fingers.

The fact that puncture with a needle in the domain of anæsthesia of the surface is very often unattended with escape of blood has been repeatedly remarked and described. Pitres attributed this appearance to an abnormal mechanical irritability of the cutaneous vessels, which is seen also in hysterical patients in other non-anæsthetic regions. In certain hysterical patients a stroke of the finger-nail or of a pencil will bring out a red line which may extend itself at once to a broad red stripe. The same thing has been observed in other diseases. In many

cases there develops in the middle portion of the red stripe a pale red lash-like elevation. In these cases the most curious reliefs can be traced upon the skin, a condition which Dujardin-Beaumetz and Mesnet distinguish as "autographism." But this autographism is not confined to hysteria. Epilepsy sometimes shows the same condition in the most exquisite form. Hysterical autographism may persist for years.

Local asphyxia has been observed during the sleep paroxysms of hysteria by Armaingaud and Löwenfeld. In the patient of Löwenfeld, a woman aged twenty-nine, who had repeated sleep attacks during the day, there developed on the fingers and nose changes very like the trophic changes which the French call the "blue œdema of hysteria." The fingers looked as if they had been frozen. The backs of the fingers were livid, and also part of the nose showed the same alteration. During an attack the fingers, including the bed of the nails, assumed a very dark blue color. After a time the ring finger of the left hand distinguished itself from its cyanotic neighbors by a remarkable bright red color, but it was, like them, swollen and cold. Still later it was observed in some fingers that the joints became cyanotic, while the remaining parts were colored bright red, so that these fingers were encircled with narrow cyanotic rings with brighter crimson borders.

The subcutaneous and even free hemorrhages which occur from unbroken surfaces furnish still more striking evidence of changes in the walls of the bloodvessels. Thus, Astley Cooper reported a case of hemorrhage from the skin of the breast after the development of an extravasation in it; Huss reported a case of hemorrhage from the skin of the scalp; Tittel saw hemorrhage in the forehead, hands, and feet and other parts of the body.

With reference to stigmatization of hysteria—that is, the occurrence of red spots with subsequent hemorrhage of the skin, corresponding to the wounds of Christ—studies with hypnosis prove that the skepticism of former times is no longer so justifiable. Thus, Faccachon was able by suggestion to raise vesicles like those of blisters on the skin; Jendrassik and Kraft-Ebing produced eschars; and Bouren, Burot, Berjon, Mabille, and others actually induced hemorrhages. Mabille found that auto-suggestion in hypnosis could bring about hemorrhage. "It is, therefore, not so surprising that certain hysterical patients who have concentrated their minds continuously upon the wounds of Christ should be able to induce hemorrhages in these regions, especially when, as was observed in the case of Louise Lateau by Boëns, the hemorrhages could be produced mechanically by mere rubbing with the fingers or with a rough cloth" (Löwenfeld).

But the belief that all this change depends upon muscular contraction and dilatation is erroneous. Much of it is the action of the cells themselves which compose the wall of the capillaries. These cells may swell and shrink like other independent protoplasm, the amœboid body, or white corpuscle of the blood, and thus increase or decrease the calibre of the capillary tube. Thus, Biedl found that the injection of a warm 0.6 per cent. solution of common salt produces a remarkable contraction of the vessels. This contraction occurs with great certainty in capillaries, arteries, and veins as a result of increased thickening of the walls.

It could never be seen that it was the result of a contraction of circular muscular fibres, as there was never any corrugation or wrinkling of the intima. The wall of the vessel becomes thicker, while the optical appearance of the interior is as smooth as that of a dilated vessel. The condition, therefore, is not that of a contraction or shrinkage, but of a thickening of the vessel wall, a so-called vital phenomenon. When the solution of common salt was washed away the wall became thinner and the lumen larger.

Enough has been cited to show that the bloodvessels are not inert tubes, but are highly differentiated structures concerned with the most intimate processes of life.

ACUTE AORTITIS.

Acute and Subacute Arteritis.

Acute Arteritis.—Acute inflammation of the walls of the arteries has been observed by the older pathologists under the general signs of sepsis, and suppurative processes in the walls of the vessels attended by coagulation of blood in the interior have been seen, especially in connection with the acute infections, typhoid fever, pneumonia, influenza; also in variola, scarlet fever, and diphtheria. The disease process begins in the vasa vasorum. The subacute and chronic inflammations were observed more frequently in syphilis.

This acute arteritis may lead to entire occlusion of the vessel, with necrosis of tissue, gangrene, or may by discharging infected matter disseminate metastases and develop septicæmia.

Acute Aortitis.—Inflammation of the wall of the aorta receives separate consideration, especially by the French pathologists, Lancereaux, Huchard, and Potain. Within the past few years Aufrecht and Bäumler of Germany and Sansom of England have isolated the lesion with distinct symptomatology. The disease is ushered in with dyspnœa, marked especially, according to Sansom, with long and painful inspiration and short expiration and with tendency to orthopnœa. A sense of constriction in the throat deepens into pain, which sometimes becomes so severe as to approach the excruciating agonies of angina pectoris. Traube had called attention to dilatation of the ascending aorta and elongation to such extent as to displace the heart to the left, and Faure and Curschmann observed the prominence of the arch of the aorta in the neck. The subclavian arteries are displaced upward, so that they may be felt above the edge of the clavicle (Potain).

The TREATMENT is the same as that of acute endocarditis. The patient is to observe rest. Pain is relieved by the application of the ice bag, which should be placed over the manubrium sterni. Sansom especially recommends the use of a liniment of belladonna, aconite, and oil of peppermint, equal parts, mixed with a quantity equal to the whole of benzoated lard. The creamy ointment thus formed is to be rubbed gently into the painful area by a pad of wool or applied upon a piece of lint. More severe pain calls for the use of opium. Digitalis

is dangerous. The dyspnœa may be relieved by inhalation of amyl nitrite or ethyl iodide. These remedies, especially the ethyl iodide, which is unattended with the uncomfortable sense of fulness in the head caused by amyl nitrite, may be inhaled in dose of five drops from the palms of the hand held in a cone over the face. Subacute cases becoming chronic are benefited by the iodides, according to the method mentioned later.

Chronic aortitis is described under Arterio-sclerosis and Atheroma.

ARTERIO-SCLEROSIS.

Synonyms and Subdivisions.—Arterio-capillary fibrosis; Aortitis; Sclerosis of the coronary arteries, of the pulmonary artery, of the arteries of the kidney, of the brain, etc.

Arterio-sclerosis (*σκλήρος*, hard) is a hyaline degeneration of the structural elements of the arterial wall, with connective tissue substitution, hyperplasia, and subsequent contraction with induration, whereby the characteristic functions of the vessel wall, retention, absorption, metabolism, with the power of expansion and contraction, are weakened and lost. The hyaline process is usually associated with atheromatous (*αθήρη*, grumous) and often with chalky and fatty change.

Atheroma and arterio-sclerosis, though apparently different, are manifestations of the same pathological state. Atheroma, which is a lesion of the great vessels, is really only a clinical manifestation of arterio-sclerosis of the vessels (vasa vasorum of these vessels). Arterio-sclerosis is the essential primitive lesion; atheroma is the secondary lesion (Oettinger).

Where does the first change begin? Is it in the blood or in the bloodvessel wall, or is the very first change in the nervous system? The blood itself is no longer regarded as the changeable fluid of former times, continually consumed and reproduced. Many of its elements, many of the blood corpuscles, for instance, persist during the whole life of the individual. Whether the changes of disease or of age begin in the nerves, in the blood corpuscles, or in the bloodvessel walls cannot be determined as yet.

Eichhorst, Rosanoff, Wassilief, describe the influence of the nervous system in trophic effects upon the artery. Giovanni cut the trunks of the great sympathetic in the neck of a dog and observed later atheromatous spots in the arch of the aorta and in the descending aorta. Botkin found endarteritis frequently after paralysis, and Huchard observed pronounced atheromatous alterations in the arteries of the arm of a man who had previously suffered from brachial neuralgia. Lancereaux goes so far as to consider atheroma as, above all things else, a trophic lesion of the nervous system. Nordman found 9 aortic lesions in 100 cases of tabes. Of 130 cases of heart disease in tabes, 51 were cases of aortic lesion (38 insufficiency, 7 stenosis, 6 insufficiency and stenosis), 4 aneurysm, 10 aortic and mitral lesion, 33 mitral lesion alone. The

other cases seemed to be chiefly myocarditis. Symptoms which recalled angina were seen in 23 cases. Usually, however, the relation is reversed and the change in the nervous system is ascribed to atheroma or arterio-sclerosis. The genesis of the process is discussed later.

Wherever or however it begins, certain it is that the process in its onset and throughout its course is, as a rule, insidious. It begins to show itself in the bloodvessel walls at or before the full period of maturity of the individual. To be able to recognize the very first changes would be to lift the veil which separates the known not only from the unknown, but from the as yet unknowable.

Individuals differ much as to the time of occurrence and degree of this degeneration. It is sometimes marked at a period as early as the age of thirty-five; on the other hand, certain individuals reach advanced age without developing arterio-sclerosis. Perhaps it would be more true to say that individuals reach advanced age because of the absence of arterio-sclerosis, for, as will be seen, most deaths are due directly or indirectly to this degeneration. Hence the common saying, first by Cazalas (1849), is true—that "A man is as old as his arteries."

The process is variously diffused in the body. The vessels under greatest strain are said to suffer first. According to Rokitansky, with whose statements agree those of Lobstein and Curci, who examined 116 cases, arteries are affected in the following order of frequency: ascending aorta and arch of the aorta, descending aorta, splenic artery, iliac and crural arteries, coronary arteries of the heart, internal carotid and vertebral arteries in the skull, uterine arteries, internal spermatic arteries, common carotid arteries, hypogastric arteries. The most rarely affected are the coronary arteries of the stomach, the hepatic arteries, and the mesenteric arteries. These statements refer, of course, only to large arteries. In the smaller and smallest vessels it is also seen that certain vessels are much more distinctly affected than others. Thus the vessels of the skin and the subcutaneous tissue, the vessels of the serous membranes, and the vessels of the kidney suffer most.

The process often coincides with the evidence of sclerotic, atheromatous, or chalky degeneration elsewhere. Fatty degeneration of the upper segment of the cornea is so commonly found in advanced age as to be known as the *arcus senilis*. The sclerotic process in the vessels initiates the sclerotic process in all the organs, which leads to the atrophy of age.

It is a curious fact that the degenerative process may be strictly limited. Thus the radial, the temporal, or the thyroid arteries may be alone affected. There may be extreme alterations in the arteries of one organ, and but little in those of another. Sometimes there is observed an extensive general atherosis of the whole aorta, as well as of the arteries of the extremities, with perfectly intact coronary arteries. Sometimes the reverse is observed. Sometimes the aorta is affected in its whole extent, while the arteries of the extremities may show only intimations of the condition, or the arteries of the brain may be markedly sclerotic, while the aorta is entirely free. The condition may be localized in the kidneys to produce the cirrhotic kidney. Cirrhosis of the liver is a disease apart. The cerebral arteries may be affected alone or in association with arterio-sclerosis elsewhere. Most unfortunate

are the cases of affection of the coronary arteries or of circumscribed rings or patches in the aorta which occlude the orifices of the coronary arteries.

Under staining with polychrome or methylene blue it is seen that individual spots in the connective tissue and musculature take up the stain more distinctly than the rest of the wall of a vessel, showing a more extreme alteration at these particular parts.

PATHOLOGICAL ANATOMY.—Hyaline (ὑαλος, clear) degeneration is the conversion of the vessel wall into a clear, transparent, homogeneous mass with destruction of all characteristic elements. The process is thus described by Ziegler: In hyaline degeneration the wall of the vessel becomes thicker and loses its lumen, so that the vessel is converted into an extremely narrow tube whose outside surface is deformed by irregular protrusions. Finally, the lumen may be closed and the endothelial nuclei, which were at first unchanged or may even have been exuberant, disappear. In other cases the hyaline substance is deposited in drop-like formations only in the vicinity of the vessels, without narrowing their lumen. Such changes are especially observed in the vessels of the glomeruli of the kidneys, of the thyroid gland, and of the brain; also of the lymph vessels, of the choroid, and of the retina (Oeler); in the last place in lead-poisoning.

It may not be said definitely how this hyaline process originates, but one gets the impression as if the vessel wall was permeated by a fluid which then sets. It is probable that colorless blood corpuscles and blood plaques may furnish the material for the hyaline substance. According to Oeler, the red blood corpuscles take part in the process. The specific tissue elements in the domain of the degeneration, as well as the cells of the connective tissue, suffer a continued atrophy. The connective tissue becomes uniformly homogeneous at first and loses its striation. Later are formed masses by separation, as in amyloid change.

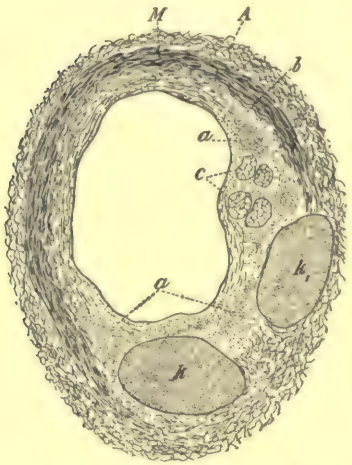
In appearance hyaline degeneration resembles amyloid degeneration; in fact, part of the hyaline masses may pass over into amyloid matter, so that combinations of the two degenerations occur: it resembles it also on account of its seat in the connective tissue and in the blood-vessels, but does not show its peculiar reactions. In every case it is a matter of the most exquisite regressive alteration.

The cause of the hyaline degeneration is entirely unknown. The characteristic feature is the resistance of hyaline vessels to acids and alkalies. Ernst has lately suggested a color reaction which is claimed to be characteristic of hyaline change. The coloring consists of a mixture of acid fuchsin and picric acid, which imparts to hyaline matter an intensely red color. Carmine and eosin likewise color hyaline matter.

Atheroma.—With the hyaline are associated the atheromatous, chalky, and fatty degenerations. Atheroma shows its effect mainly in the intima. The smooth flat cells which compose the lining membrane of the intima are converted into thick, opaque, yellowish white plaques, which thicken and roughen the surface. Whitish and yellowish spots appear at first scattered about the surface, protruding from the surface or visible apparently in the depth of the tissue. These spots coalesce to form larger and larger surfaces, so that the

whole interior of the aorta may be finally lined and roughened with yellowish white sclerotic tissue. So long as there is no solution of continuity the vessel, though it will have lost contractility and resilience, offers no obstacle to the circulation, but the atheromatous matter is liable to softening and necrosis. With the disintegration of this matter detritus, fatty matter, cholesterine, and fibrin from the blood are deposited upon the surface to constitute the atheromatous—*i. e.* gruel-like—ulcer. The atheromatous change in the aorta was first of all described by Hodgson (1815), who noticed the calcareous and pultaceous condition of the lining membrane, and called attention to the dilatation of the aorta beginning above the semilunar valves. The accompanying plate represents in cross-section the changes of atheroma. (See Plate II.)

Fig. 24.



Atheromatosis of the mesenteric artery: *A*, adventitia; *M*, muscularis. The intima shows at the top fibrillary, at *a*, a small-celled infiltration; at *c*, cavities from absorption; k_1, *k*, chalk deposits.

This caseous and fluid disintegration of tissue gave rise to the term atheroma. Corresponding terms were the "pulticula" of the Romans, the "bouillie" of the French, and the "pap" of the old English writers (Sansom). In the well known case of the sculptor Thorwaldsen this sudden death was found to be due to the fact that an atheromatous ulcer had broken into the coronary artery and had filled and blocked the artery with a soft, putty-like mass.

The edges of the ulcer may be sinuous or may be overlapped, so that the probe may be inserted some distance beneath the intima. In other places the intima is broken by fissures and is more or less universally roughened. In consequence of the increased resistance from the action of the heart the vessel becomes elongated, serpentine, dilated into aneurysms, and so friable as to suffer rupture. The thickening of the intima is often so great as to lead to thrombosis, occlusion, endarteritis obliterans.

Calcification.—A further degeneration which may now ensue is that of calcification: deposits of chalky matter, which aggregate to form plates, substitute the natural tissue and convert the vessels into bony or stony tubes, which have lost all power of expansion, contraction, elasticity, and response to nervous influence. Chalky deposit is rarely entirely uniform, but is usually interrupted or broken, so that medium sized vessels, like the radial and temporal, feel like a chain of beads or like a long slate pencil much fissured and cracked across. The interior may be at the same time so much roughened as to lead to the development of thrombi which may firmly adhere to the surface and finally block the vessel (arteritis obliterans), or may, by being swept by the circulation to distant points, come to lodge as emboli. It is interesting to know that emboli thus formed may become organized, and may sub-

PLATE II

Atheroma of the Arch of the Aorta, also of the Ascending Aorta Implicating the Valves to Produce both Absolute and Relative Insufficiency. (Lehmann's Atlas.)

sequently in turn undergo the same degeneration, especially calcification (Marchand).

However produced, the process begins as a deposit of minute, highly refracting granules which gradually coalesce. The process is for the most part a pure calcification. It is a curious fact that in certain cases a real ossification, with bone lamellæ and corpuscles, may take place in the media. Marchand mentions such a case in an organized thrombus in the crural artery, and Cohn reports three such cases.

The amyloid degeneration begins in the small arteries, and extends thence to the capillaries and the circumjacent connective tissue. The process begins as vitreous lustrous swellings in certain parts of the vessel wall, while the intervening parts remain unaffected. The affected parts gradually coalesce, so that the whole vessel becomes uniformly involved. The amyloid substance is deposited chiefly in the media, and in the process of thickening so greatly contracts the lumen of the tube as to produce anæmia of organs in the domain of the circulation. The amyloid degeneration is known by its characteristic color reaction with iodine, sulphuric acid, and methyl violet. Bloodvessels which have suffered amyloid change are converted into inert tubes, readily permeable, though not so resistant to the influx of blood as in hyaline or chalky change.

Fatty Change.—Simple fatty degeneration of the intima is very frequent, but is usually slight. The process develops small round or oval, slightly elevated spots of whitish or yellowish color, which are especially abundant on the inner surface of the aorta just above the valves, and next on the posterior aspect between the orifices of the intercostal arteries. Such spots occur also on the valves. The surface of these spots is usually smooth, but sometimes slight roughness is formed by superficial necrosis, to constitute the "fatty usury" of Virchow. The process occurs at all ages, and is particularly pronounced in the anæmic and chlorotic. Fatty degeneration occurs also in the walls of the small arteries. In the media the process develops with a fatty destruction of the muscle fibres along with atrophy. The elastic layers suffer likewise, partly under influence of the continued pressure and partly under extension of the necrotic process.

Under the microscope the wall of the vessel is seen to be decidedly thickened, the intima often as much as three or four times. Fat globules infiltrate the whole tissue. The polygonal cells of the endothelium show signs of fatty degeneration. The calcified surfaces exhibit a more homogeneous appearance, as the process destroys the cellular elements with their nuclei. The media likewise show signs of atrophy, fatty and chalky degeneration, so that the muscle fibres can be recognized only with difficulty or not at all. Finally, the characteristic elements of the elastic tissue itself are lost.

Periarteritis.—A peculiar degeneration occurs in the form of a periarteritis, in the shape of nodules which represent inflammatory dépôts deposited upon the smaller arteries, as first described by Kussmaul and Meier. These nodules were found by Fletcher and Kahlden to consist of exuberations of endothelium which penetrate the media and induce a small celled infiltration of the adventitia. The weakening of the wall of the vessel may give rise to aneurysmal dilatation, or the thickening

of it to the occlusion of the lumen by thrombosis. The cause of the condition is entirely obscure. The nodules have usually been encountered in weak, anæmic individuals suffering especially from nephritis, gastro-intestinal disturbance, pains in various parts of the body, especially in hypochondriacs with a quick pulse and a low subfebrile temperature. The disease is very grave. Patients succumb by marasmus in the course of six weeks to three months. In a case reported by Kussmaul the nodules could be felt through the skin—a point which may be of value for diagnosis in the future.

Beginning of Arterio-sclerosis.—Again, the question forces itself to the front, Where does the process begin? In the vessels, in the blood, or in the nervous system?

The attempt is made to explain the whole process in a mechanical way. Thoma, for instance, ascribes the diffused thickenings of the intima to abnormal distention of the media, which in turn depends partly upon reduction of contractility and partly upon increased resistance at the periphery. The fact, however, that the condition is seen so often in earlier life as the result of infectious processes (rheumatism, syphilis), and may be at any time precipitated by certain poisons, especially by alcohol, lead, or uric acid, would indicate the existence of some toxic agent in the blood. And, in fact, the modern tendency is to locate the origin of the condition in the blood.

Ziegler lays stress especially upon the fact that the arterio-sclerotic process is attended with the evidence of inflammatory infiltration, especially in the walls of the vasa vasorum, so that in essence arterio-sclerosis is an arteritis prolifera or hyperplastica, which is described as an endarteritis, a mesarteritis, or a periarteritis according to the part of the vessel which is first or most affected. Obliteration of the vasa vasorum leads in turn to atheromatous degeneration of the vessel.

The vasa vasorum certainly show alterations throughout the whole process. Numerous round cells, leucocytes, escape from their interior and penetrate to the tissue of the intima. Connective tissue multiplies, partly by transformation of these bodies, and leads eventually to the formation of cicatricial tissue.

Burchard found in 6 cases of spontaneous gangrene of the extremities more or less complete occlusion of the artery by an obturating mass which consisted of lightly striated connective tissue with spindle shaped or more oval endothelial-like cells. These cells were not uniformly distributed, but were collected in crowded groups and in places passed over into the old epithelium. These obturating masses were surrounded on the outside by a homogeneous layer poor in cells. This was the picture in all the cases where the disease was advanced, and either all the arteries of the extremity were uniformly involved or it was only in one of them that the disease showed itself. Usually it was only the chief branch which was affected up to its periphery, while the smaller branches and the capillaries remained free.

Köster declares that every hyperplasia, whether due to simple atheroma or to syphilis, as of the brain arteries, depends upon mesarteritis, which reaches the vessels from without through the avenue of the vasa nutritia. In every endarteritis and arteritis the chief rôle is played by the vascular apparatus, and the exuberation of the intima is not de-

pendent upon the endothelium. An endarteritis can occur, therefore, only in vessels which are supplied by vasa nutritia, as Cohnheim had declared that individual parts of the body, including or considering the vessels as organs, can be the seat of an inflammation only when they possess vasa vasorum.

Hollis finds in the earliest stages of atheroma corpuscles in the endothelium and basement membrane which could be easily stained with basic dyes. These bodies are regarded as vagrant corpuscles from the blood determined to the particular region in consequence of the invasion of foreign matter, the passage of which in the form of particles into the minute arteries which form the blood supply of the coats of the vessel constitutes the first step in the disease process. Hippolyte-Martin makes the process more mechanical, and considers the changes and obliteration of the end arteries the result of occlusion of minute branches of the vasa vasorum.

Fränkel would put the genesis farther back in the nervous system. In two cases of mal perforant in tabes he removed the second toe and studied it anatomically. The most obtrusive thing was the extreme hypertrophy of the media in the finest arteries. Fränkel had seen the same thing in the tibial nerve in the case of a leg amputated for spontaneous gangrene and in the sciatic nerve of a tabetic patient. Hereupon were studied 36 different cases of diseases of the nervous system (tabes, multiple neuritis, progressive paralysis, syringo-myelia) as to the condition of the peripheric arteries, mostly the anterior tibial artery, and in all the cases the same conditions were found. There were usually two types: one consisted in hypertrophy and hyperplasia of the media without dilatation of the vessel, the other in a general increase in the size of all parts of the vessel, with hypertrophy of the media and intima. Chalk deposits were often found in both layers. Analogous conditions were seen in the veins.

The changes of age play no rôle in these cases, so that the suspicion was excited that the trophic changes which caused the disease were developed from an angio-sclerosis. In order to study the question experimentally, Fränkel cut through the sciatic in three dogs and two rabbits. Hereupon the most fiery trophic changes set in at once, and the anatomical study after death of the animals showed changes in the vessels of the same character as those described. Fränkel thinks, therefore, to have furnished proof that the so called trophic changes in the vessels are brought about in an indirect way through lesion of the nerves, and proposes the name neurotic angio-sclerosis.

The frequency of affection of individual arteries points to some local cause. The rôle of a mechanical factor has been especially emphasized by Traube, who attributes sclerosis to retardation in the rapidity of the current. The fact is, however, that the arteries most often affected are those in which the blood current maintains its original velocity in the closest vicinity of the heart. A more important element is increase of arterial pressure, and perhaps a still more important element is a frequent marked change in the pressure. The great frequency of affection of the aorta points to a mechanical effect of the blood pressure upon the vessel wall (Marchand). But, not in any way to strain a point for proof, what is there in any evidence of this kind to exclude the action

of some toxic matter which shows its effect in the aorta, in cerebral vessels, and in certain capillaries, as in the well known lesions of syphilis, rheumatism, and gout? Thus, though Krabow and Lubarsch could not succeed in causing amyloid degeneration by inducing chronic suppuration in dogs, Czerny caused it in five cases, especially in the spleen, by long continued injections of turpentine.

Miller (Edinburgh) attributes the muscular atrophy of tuberculous joint disease to contraction of the chief bloodvessels and insufficient nutrition. The author starts with the statement that the atrophy is too great to be accounted for by disuse, and finds explanation rather in the reflex contraction of the vessels. He claims to have seen such contraction, and ascribes it to irritation excited by products of metamorphosis from tubercle bacilli. Von Winiwarter ascribed his cases of spontaneous gangrene to noxæ in the form of colds and freezings, which constitute repeated injuries and beget in this way a chronic exuberative process in the vessels and nerves. This view is very probable, but there must have been predisposing circumstances, possibly hereditary syphilis or gout. (See Plate III.)

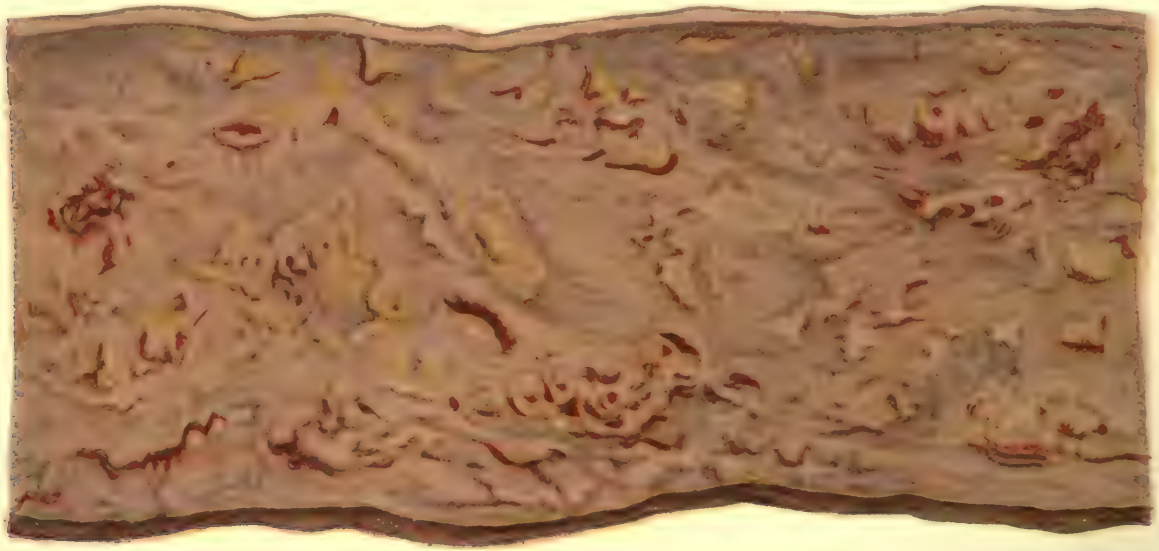
Effects of Sclerosis.—The first effect of sclerosis shows itself in impairment of contractility, later of elasticity and osmosis.

Langhaus has shown that the number of muscular layers is reduced from the normal forty or fifty to thirty or forty, and the thickness of the wall from its normal standard of 1.2 mm. to 0.4–0.6 mm. With calcification the impairment of function becomes extreme. John Hunter showed long ago that a normal aorta could be stretched to nearly double its length, and that when the tension was relieved it would return to almost its normal size. Polobetnow proved that under the same tension a sclerotic artery could be stretched much less than the normal, and Thoma demonstrated that on relief of the tension the artery remained longer. In fact, the ascending aorta and the arch of the aorta may be in life dilated to double the natural size, and thus form real aneurysms.

The next effect is the impairment of osmosis and the interference with metabolism. Hyaline matter, like most inert matter, permits the readier passage of fluids. Hence the increased permeability of the vessel walls, and hence the rapid accumulation of fluids under the skin and in the serous cavities to constitute the various dropsies, and the case of the exudation of albumin from the walls of the vessels of the Malpighian bodies. Hence, finally, the failing nutrition, emaciation, and marasmus of this state.

Again, the rapidity of the circulation stands in direct relation to tissue changes in the vessel walls. The capillary walls of many old people after death are much more permeable than those of the young. Every increase of permeability increases the resistance to the blood current. This retardation of circulation through a more or less rigid tube was first observed by the immortal Harvey in his interesting experiment of cutting an artery across and then tying a tube in it. Harvey says: "For as soon as you will have tied the artery it will begin to dilate above the quill or pipe, on account of the impulse of the blood driven from above, so as to exceed the circumference of the tube; whence the flow of the blood will be arrested and its impulse broken, so that the pulsation of the portion of the artery placed below the liga-

PLATE III.



Atheroma of the Thoracic Aorta. (Lehmann's Atlas.)

ture will be lessened, as it is not the impulse of the blood that flows through it which is driven back above the ligature."

When arterio-sclerosis is pronounced the vessel offers such resistance to circulation, and so little help to it, as to throw extra work upon the heart and lead to hypertrophy of the left ventricle. This hypertrophy must in the course of time give way to dilatation. When the nutrition is bad, as in old or enfeebled individuals, dilatation occurs rapidly. But if the heart continues strong while the vessel grows weak, the wall of the vessel is subjected to tension, sometimes to dilatation (aneurysm) and rupture (apoplexy).

ETIOLOGY.—The factors which operate chiefly in the production of arterio-sclerosis are age, sex, alcohol, syphilis, rheumatism, and gout. To these factors may be added certain acute infections, high living, and hard work.

Age.—Gruenstein finds that the intima and media increase in thickness with increasing age. In the case of the aorta, carotid and subclavian arteries the relative increase of the intima is greater than that of the media; in the common iliac artery the ratio is reversed. In the first mentioned arteries the relative increase in thickness of the intima in comparison with the media is greater in the first years of life up to adult age, while in the iliac artery the ratio remains almost constant during the whole of life. A part of the elastic tissue in the aorta, subclavian and carotid arteries is altered in advanced age, so that the elasticity of the arterial wall is reduced (Unna).

Age is a very indefinite expression. Some men are old at forty; some men are young at sixty. According to comparative anatomy or zoölogy, man is entitled to live one hundred years. The actual fact is, however, that life is much abbreviated, mostly by disease and mostly by this disease. Aside from accidents, under which may be included deaths from the acute infections, life is usually terminated, as stated, by the slow process of arterio-sclerosis. Advancing age is commonly said to produce this change by failure of nutrition. This statement is, however, only a paraphrase: it is a failing of the nutrition which produces the signs of old age. The subject may be set a little farther back by accepting the statement that the strength of the arterial wall—that is, the degree of contractility—more especially the abundance of active muscular tissue, are matters of original endowment—that is, of heredity. Hæmophilia, which is a distinctly hereditary affection, is distinguished by thin walled vessels. There is no doubt that individuals differ at birth in the thickness and strength of the arterial walls as much as in the structure of other organs or features. It is accepted that the conditions which lead to arterio-sclerosis may be transmitted, and the evidence of it runs in families, as long life runs in certain families, or as premature apoplexies, heart failures, aneurysms run in other families. Huchard goes so far as to speak of an "hereditary aortism." But the influence of heredity is largely counteracted by the conjunction of the sexes, as the effect of sexual selection is manifested chiefly in securing variation.

The influence of age in the production of arterio-sclerosis is really largely a matter of habit, with reference to the conservation or dissipation of force by a life of self-denial or self-indulgence, with reference also

to protection against disease, to the ability to secure the comforts of life, etc., as "luxury and longevity are twins."

But the gross alteration of atheroma is undoubtedly a disease chiefly of middle and advanced life. Of 93 individuals so affected, Curci found 77 forty years old and over. The great Harvey reported concerning Thomas Parr, who died at the age of one hundred and fifty-two years, that he could not find any trace of alteration in the arteries, but Demange declared that in 500 autopsies of old people he never met with a single case in which there was absolutely no atheroma. The finer changes of arterio-sclerosis are seen much sooner. Thus, Guéneau de Mussy found that of 160 cases of alteration of the arterial walls in different degrees, 80—that is, just one half—occurred in subjects under forty-five years of age, and Curschmann found sclerosis of the aorta and aneurysm six times in individuals under twenty-five, and both conditions still oftener in individuals under thirty years of age.

It is remarkable that we sometimes find an entire absence of deposit in the whole aorta at the age of seventy or eighty years, and that, too, while the evidence of senescence may be present elsewhere in pronounced form. Wherefore it is that the deposits of atheroma may not be attributed to senescence as such, but to entirely local causes. The pure senile process is more general, like that which shows itself in the skin. It constitutes a sclerosis of less grade. It is to be regarded as a physiological change, not pathological. Such a conception is of great value in prophylaxis, for if the process of arterio-sclerosis be not due to age, there is always a possibility of prevention (Bäumler).

Arterio-sclerotic changes sometimes show themselves in children with weak intellects in early youth. Leubuscher reported the case of a feeble-minded, extremely nervous boy aged fifteen, in whom there was a distinct and pronounced arterio-sclerosis of the temporal artery, though changes in the arteries could not be demonstrated in other parts of the body.

Sex.—The male sex suffers most, for the simple reason that men are more frequently affected with alcoholism, syphilis, gout, etc., which directly induce the degenerative change; further, men work harder than women, as a rule, and thus subject the arteries to greater physical strain. We are much in need of more accurate observation upon this question of sex. In a collection by von Basch of 398 cases of partly latent and partly beginning and advanced sclerosis it was seen that hardening of the vessels was nearly as frequent in fat persons as in lean, and that very fat persons often show a permanent low pressure. It might, therefore, be believed that the same causes may favor on the one hand arterio-sclerosis, and on the other an excessive increase of fat.

Alcohol acts in every way. In the first place, it over-excites the heart, so that for a time at least more blood is pumped into the vessels and the vessels are subjected to greater strain. Secondly, alcohol disturbs the action of the digestive organs, produces gastric catarrh, and especially interferes with the function of the liver. Alcohol exercises especially deleterious influence upon the liver. The stay is longer in the liver. It can be obtained in larger quantities from the liver than from any other organ in the body. The capillary system in the liver is peculiarly rich and delicate; the whole gland finally suffers in cer-

tain cases the characteristic atrophy distinguished as cirrhosis. It is a principal function of the liver to eliminate poisons with the bile and empty them into the intestinal canal. Disturbance of the function of the liver loads the blood with these poisons, and they in turn act upon the bloodvessel walls, especially through the medium of the vasa vasorum. It is not improbable that the ultimate cause of so-called idiopathic or autochthonous arterio-sclerosis will be found in waste products (auto-intoxication) that should be eliminated from the body.

The action of alcohol is by no means simple. It is not so much the irrigation of the walls of the vessels with alcoholic blood that does the damage; it is the effect of alcohol upon the radicles of the portal vein, upon the hepatic veins, and upon the pulmonary artery and its branches. The fact is, that we sometimes see in notorious drinkers but scanty deposits in the aorta and other large arteries, but abundant lesions are encountered in the parenchyma of the liver, the kidneys, and the brain. The destruction of these parenchymatous cells leads to secondary proliferation of connective tissue, which extends to the small vessels of the affected organ and produces a progressive sclerosis to constitute the obliterating endarteritis. In the case of a highly vascular organ like the kidney the contraction of vessels has an effect upon the whole circulation, especially upon the heart. Thus is developed a gradual insufficiency of the kidneys, which loads the blood with new toxins (Bäumler).

Finally, alcohol is itself, when taken in excess—that is, beyond the capacity of the emunctories—a poison to the blood. Alcohol is a direct toxic product of a vegetable micro-organism. The reason why alcohol does not show more general diffuse sclerosis is that it usually cuts life short by disease of individual organs, especially of the heart. Of course the danger, aside from the quality, depends upon the amount. In moderate quantities alcohol may support a failing heart. It is true that "wine is the milk of old age," but any excess wears out the heart, and poisons it and its vessels besides. Nevertheless, with all this a priori reasoning it is only fair to state that there are authors who disclaim any connection between alcohol and atheroma. Thus, Lancereaux refuses to admit alcohol as a cause, maintaining that alcoholism more frequently produces fatty degeneration than atheroma. Duclos, based upon a large number of autopsies, believes that alcoholism should be excluded. But most of the authorities consider the influence of alcohol as unmistakable. Dogs that have been subjected to long continued alcoholic intoxication show the same degeneration of the aorta. The fact that many confirmed drinkers show no lesion at all seems to speak against it, but the frequency of sclerosis in young people the subjects of alcoholism points decidedly to the relation of cause and effect.

Syphilis has long been recognized as an important factor in the production of disease of the artery walls, especially in the production of aneurysm. Heubner called especial attention to an affection of arteries of the brain entirely due to syphilis.

The effect of syphilis is shown in reports from military life. In 114 autopsies on soldiers Davison found atheromatous lesions in 22 cases, of which 17 had had undoubted syphilis, while in 78 who had not had syphilis only 4 presented the same lesions. Huber reported a typical case in a young man but twenty-two years of age.

Syphilitic arteritis may be diffuse or circumscribed. The disease has predilection for the basilar arteries, which are rendered rigid and show throughout their course spots of yellowish white thickenings, with corresponding diminution in the lumen of the vessel. The extension of the inflammatory process agglutinates the wall of the artery to the tissue of the pia mater, and through it to the substance of the brain, which may show numerous spots of cicatricial tissue or dépôts of softening or gummatous products. The intima in these cases undergoes so much thickening as to lead to great diminution or final obliteration of the vessel (endarteritis obliterans), as illustrated in the accompanying plate. Gummatous nodules may form also in the adventitia, where they may closely resemble tubercles.

FIG. 25.

Syphilitic arteritis; endarteritis obliterans. The greatly thickened intima shows partly fibrous and partly small-celled infiltration.

In the studies of Charrier and Klippel under the direction of Dieulafoy it was observed that syphilitic affection of the brain arteries shows changes which are partly to be regarded as specific and partly as not different anatomically from the condition produced by other diseases. The specific affections are, on the one hand, secondary, produced by dépôts in the vicinity; on the other hand, primary, with their seat in the wall of the artery. The authors studied especially these later changes, and distinguished the gumma of the artery wall and the syphiloma which had become transformed into a sclerosis. The gumma develops from a proliferation of the endothelial cells. The authors were not able to observe hypertrophy of the elastic fibres, of the fenestrated membrane, or of the muscle fibres of the tunica media. Retrograde changes set in later with fatty degeneration, contraction, calcification. These changes result in contraction, occlusion by thrombi, rupture, aneurysm. It is rare that the syphilitic nature of diseases of the arteries can be distinguished from the anatomical picture. The diagnosis is supported by the fact that syphilis affects by preference certain arteries of the brain, the fossa of Sylvius, the circle of Willis, etc.—that the syphilitic affection is usually circumscribed, is often symmetrical, and is largely confined to the intima. It is observed that syphilis especially attacks the brain which has been weakened by other injurious influences. Syphilis produces, besides the endarteritis obliterans, a circumscribed granulation tumor in the arteries and veins known as a syphiloma. These tumors, in contradistinction to those of sarcoma and carcinoma, consist of small nodules which occur in the smaller and smallest arteries. It attacks the brain vessels by preference, because syphilitic arteritis is localized in arteries supplied with lymphatic sheaths, and this is the case in the cerebro-spinal arteries; other vessels,

notably the large vessels, as the aorta and its branches, are not at all affected (Lancereaux).

Acute and Chronic Infections.—Arterio-sclerosis and atheroma are observed with great frequency in individuals considered arthritic or rheumatic. Guéneau de Mussy remarked on the frequency of atheroma in chronic rheumatism; he saw it 67 times in 208 cases—over 38 per cent. May it be, Oettinger asks, that the atheroma and rheumatism are produced by the same cause?

Arterio-sclerosis is observed also with great frequency in gout, saturnism, and diabetes, where it is the cause of the symptoms, renal atrophy, lead palsy, cardiac troubles, angina, etc., seen in these diseases.

According to Huchard and Curschmann, certain acute infectious diseases may lead to arterio-sclerosis, especially typhoid fever, malaria, influenza, scarlet fever, and diphtheria. The frequent occurrence of arterial thromboses in the course of some of these diseases, which could not be derived from anything else than disease of the vessel walls, seems to speak for the possibility of such a connection.

To prove the rôle of the infectious diseases in the production of arterio-sclerosis, Thérese undertook to produce it by injecting into the bodies of rabbits and guinea-pigs virulent and half virulent, also filtered, cultures of different bacteria, of the bacterium coli, streptococcus, and diphtheria bacillus. At various times after the inoculation this observer found in all cases the following changes: Accumulations of round cells, with perfectly intact endothelium in the lumen or in the neighborhood of the wall of the capillaries of the different organs. These round cells in the walls consist nearly always of masses of microphagi (Metschnikoff), while the masses in the lumen consist of macrophagi. Similar accumulations of round cells were found grouped about the central small vessels, in the large vessels, in the perivascular connective tissue, while round cells were distributed in the tissue. Sometimes an accumulation entirely surrounded a vessel. It is easy to understand that such alterations in the vicinity of the large vessels must produce disturbance in the nutrition of the vessel wall. How these disturbances lead exactly up to sclerotic change was not remarked. The accumulation of leucocytes is attributed to the chemotactic effect of the toxins. It is not unlikely that injury to the vaso-motor nerves had something to do with the changes in the vessel walls.

Hippolyte-Martin found these changes in early childhood in consequence of infections. In all the cases of childhood the changes in the vessels had been preceded by the infectious diseases, diphtheria and other forms of angina, smallpox and typhoid fever, erysipelas and other pyogenic infections, perhaps also acute rheumatism, syphilis, tuberculosis, lepra. In any of these diseases specific micro-organisms or toxins may affect the endothelium.

The discovery of sclerotic dépôts in the aorta and other arteries of old people represent deposits of the acute infections encountered from time to time during a long life. The parenchymatous organs, like the kidneys, show the same relics.

Toxins soon affect the contractility of the vessels. Something of this effect is seen in experiments with ergot and with lead, which substances may produce a protracted spasmodic contraction of the smallest

arteries, whereby the blood pressure is increased so that extra work is thrown upon the heart and large vessels. In the case of lead and other poisons there are also secondary effects. The destruction of the cells leads to secondary proliferation of the connective tissue, which extends also to involve the smallest vessels and produce a sclerosis of the finest arteries. Moreover, the destruction of these cells overloads the blood with new toxic products and interferes with the elimination of toxins.

There is no doubt that depressing psychical emotions play a prominent rôle in helping the process along.

The Relation of Food.—Fränkel, Fräntzel, and Fritsch, all recent authors, invoke a simple plethora of vessels from over-feeding and drinking, with a consequent rise of blood pressure, hypertrophy of the heart, and distention of the vessels, as a sufficient cause of the condition. This so called "luxus consumption" occurs especially in high livers of sedentary habits with tendency to corpulency or obesity. The high tension thus produced mechanically is further favored or increased by toxins from over-feeding which continually circulate in the blood in these cases.

The process of calcification is said to be especially marked in individuals who live exclusively upon vegetable food. Alanus observed it in himself, a young man not yet forty years of age and not addicted to alcohol, in the temporal and radial arteries. Raymond saw it in a number of young monks, vegetarians, and Treille remarked it of the inhabitants of Bombay and Calcutta, who live largely on rice. According to Garber, the mineral salts in the vegetables come to be deposited in the walls of the bloodvessels: "Vegetable food injures the bloodvessels and precipitates the changes of age." This statement needs confirmation.

Drinking water saturated with lime salts, with vegetable food, has been accused of producing sclerosis (also by Gubler), but that a predominant vegetable diet may not have this effect is shown in the fact that in the West Indies the arteries of old people are found to be remarkably free of arterio-sclerosis (Stevens).

Hard work acts by raising the blood pressure and subjecting the heart to hypertrophy and the vessels to strain. Josserand observed arterio-sclerosis, especially the arterio-sclerotic kidney with secondary affection of the heart and lungs, remarkably frequent among the silk-workers in Lyons. The degenerative change is to be attributed to the hard work. It is found that the women here suffer more frequently from sclerosis than elsewhere, because, besides the work which they do all day with their husbands, they discharge their duties as wives and mothers at home.

So, arterio-sclerosis is found in all classes of society, and especially in the two extremes. But both these factors, food and work, may operate by turning toxic agents into the blood. Thus, Rosenbach finds in certain peptones distinctly poisonous properties, and Roger showed in his experiments that the blood of an animal overdriven (surmené) had such high toxic power that fifteen milligrammes of it would suffice to kill one kilogramme of the same animal. It is claimed, finally, that exposure to excessive heat, as in furnace-feeders, bakers, moulders, may lead to changes in the nutrition of the vessel walls (Semmar).

Symptoms.—Arterio-sclerosis may be extensive, as stated, and show

no external signs whatever. Thus, Floersheim reports the case of a woman affected with syphilis and alcoholism who died suddenly with symptoms of genuine angina pectoris. The autopsy revealed marked calcification of the aorta, with narrowing of the orifices of the coronary arteries. The myocardium was apparently sound. Beadles reported a case of a man aged forty with dubious syphilitic history, who suffered from sclerosis which was exactly limited to the thoracic portion of the aorta. The left subclavian and nearly all the intercostals were plugged at their origin with thrombi. Notwithstanding these lesions there were no subjective manifestations on the part of the heart. Death was caused by cancer of the tongue. On the other hand, the disease may be marked in the external vessels, and the presence of the condition be distinctly indicated by the hardness of the radial, temporal, or other artery accessible to the touch. Thus these arteries may, as stated, feel under the finger like bony tubes, pipe-stems or chains of beads, and show from elongation a distinctly serpentine course.

V. Basch distinguishes as a latent arterio-sclerosis a condition which reveals itself by increase in the average blood pressure, measured with the sphygmometer. This condition is to be separated from the arterio-sclerosis which is anatomically demonstrable and which shows itself in distinct clinical signs. This observer believes that the increase in the tension is the expression of an already existing, but not yet demonstrable, change in the bloodvessels, while Traube and Huchard look upon the increased tension in the pulse only as as an exciting cause of arterio-sclerosis. V. Basch considers any case in which the pulse tension is permanently raised above 150 mm. Hg as suspicious of arterio-sclerosis.

Coarse changes in the pulse may be recognized with sufficient accuracy with the finger, but much finer changes may be appreciated by various instruments devised for this purpose, as the arteriometer and the sphygmograph. For instance, it is noticed that a change of posture is attended by an alteration in the calibre of the pulse. This alteration in the radial artery may be as much as 25 per cent. The postural variation becomes less marked with advancing age under the changes of arterio-sclerosis. According to the observations made by Oliver with his arteriometer, the radial calibre measures in the recumbent posture 1.5 mm.–1.9 mm.—in the standing posture 2.5 mm. That is, the vessels are dilated under effort or exercise, and in this way a greater amount of blood is supplied to the muscles, to the brain, etc. These postural variations, however, as noticed, for instance, at the radial artery, may be diminished from various causes, as from contraction of the arterial wall under the influence of cold, from lowering of the action of the heart, or increased as from increase in the action of the heart.

The most common single cause of persistent uniformity in the vessels is gout, but arterio-sclerosis from any cause, especially from acquired syphilis and chronic nephritis, plays an important rôle. The curious condition known as myxœdema acts in the same way.

Oliver observed that the arteries were contracted in from 25 to 50 per cent. in a large number of cases of chronic syphilis which came under his observation. So uniform is this fact that it enabled him to detect, he says, over and over again, evidence of syphilis in the

arteries when no other clinical clue existed and when the early history of infection had been unrecognized or forgotten.

Then, again, arterio-sclerosis may be latent in the body for a long time, and the symptoms which eventually appear will depend upon the organ which is affected. Arterio-sclerosis may be widely diffused and may exist in pronounced form in the entire absence of symptoms, provided the life of the individual can be adjusted to the altered conditions of the circulation. The general reduction of energy which goes along with atrophy of organs in advancing age naturally adjusts the demands of the body to the failing supply. The individual learns, or is soon taught, to economize force. As the voyage of life advances he realizes that, as Emerson said, "it is time to take in sail." The conditions of life, however, do not always permit such an adjustment. Moreover, the process may be precipitated so early in life or may be so pronounced in individual organs as to show prominent signs. Sooner or later arterio-sclerosis manifests itself by distinct symptoms peculiar to the part or organ affected.

Atheroma of the Aorta.—When the aorta suffers in marked degree the great arterial trunk loses its elasticity, and the whole body is more or less imperfectly fed with blood. The aorta itself may be dilated into aneurysms, which in turn produce a train of characteristic signs. Affections of the aorta may implicate the orifice of the coronary arteries, or the disease process may directly involve the trunk or main branches of these vessels to produce the symptoms of coronary sclerosis, especially heart failures, arrhythmia, stenocardia. Affection of the brachial and intercostal arteries leads to defective nutrition of the respiratory muscles, with weakening of their power; to premature ossification of the cartilages of the ribs, with defective expansion of the chest; and to emphysema of the lungs.

Where the process is general, as in the so-called arterio-capillary fibrosis (Gull and Sutton, 1872), or where it involves large organs, including the aorta, the kidneys, etc., which in the discharge of their functions or secretions naturally assist the circulation or relieve or lower the blood pressure, this important function is interfered with, and the pressure rises to throw extra work upon the heart. This condition is made manifest by hypertrophy of the heart, which in turn shows itself by—(1) increase of the impact, with dislocation of the apex; (2) accentuation of the closure of the aortic valves—that is, of the second sound at the base on the right of the sternum; (3) increase of the diameters of the heart, especially to the left. The increased power of the heart is further indicated by the abnormal hardness and resistance of the pulse in the superficial arteries. All these conditions, except the increased dulness, disappear in the later course of the affection, when the hypertrophy gives way in the course of time to dilatation.

A valuable sign manifest in some, but not in all cases, and resulting from elongation of the aorta first noticed by Traube, and wholly independent of hypertrophy, is the change in the situation of the apex stroke when the patient lies upon the left side. Thus, where the stroke in the sitting posture or on the back may be seen within the left nipple in the fifth intercostal space, it may be displaced as far as the axillary line when the patient lies upon the left side.

Atheroma of the aorta often coincides with atheroma of the aortic valves. In these cases the altered sound is propagated with great distinctness through tubes with most solid walls; for the distance to which murmurs or tones can be transmitted depends not only upon their character and intensity, but also upon the resonance of the arterial connecting tubes. When the innominate and right carotid are atheromatous, one may hear the second sound ringing in the vessels affected with atheromatous processes as distinctly as over the aorta, and louder than in the left carotid, which is not affected.

And so also in predominant localization of atheroma in the left carotid the ringing sound of the second tone may be heard more distinctly in this vessel than in the right carotid—a condition which in connection with a palpable hardening of the left carotid may indicate a lesion of the artery of the Sylvian fissure, and thus have premonitory significance. If the atheromatous process in the thoracic aorta involves a greater part of it and extends to the abdominal aorta, the diastolic tone may be heard a good distance on the back of the chest at the point corresponding to the course of the aorta. These manifestations may be so distinct as to lead to a false diagnosis of aneurysm of the abdominal aorta (Neusser).

Sometimes the heart sounds can be heard distinctly in places where they are usually inaudible or feeble. Thus, it may be possible to hear the sounds distinct and tolerably loud in the left supraspinatous region, because the dilated vessels, especially at the point where the arch of the aorta begins, lie closer to the wall of the chest.

The second aortic sound is accentuated. Compared with the second pulmonary sound, it is loud and clangorous. The condition may be explained by a physical cause—to wit, by the dilatation of the aorta and the increased quantity of blood in it.

In the majority of cases, especially where the sclerotic process is diffuse, dilatation shows itself in the course of the first week, and the dilatation extends to involve the tissue about the valves. The consequence is, that this zone of the aorta is stretched and enlarged. But the valves are likewise stretched, so that, notwithstanding the dilatation, they may still perfectly close for a long time. These stretched valves necessarily furnish certain different acoustic signs from the unchanged pulmonary valves. The larger membrane produces larger and fuller sounds and gives the impression of accentuation. This peculiar accentuation is a sign of great diagnostic value, and may direct attention to the disease long before it can be recognized by subjective signs. As the dilatation increases the aortic ring becomes relatively insufficient and the signs of aortic insufficiency gradually set in (Curschmann).

Sclerosis of the coronary artery announces itself by attacks of palpitation after meals, when the vessels are unusually full, by dyspnœa after slight effort and out of all proportion to the effort, showing itself in dressing and undressing, in defecation, or in merely rising from the horizontal position. At first there is a feeling of oppression over the upper part of the sternum or a sharp pain in the left arm, which compels the patient in going or climbing to stop and take a long breath. In a few minutes he is free and can resume his effort until the condition recurs. Recurrence at first stands entirely in connection with muscular effort,

therefore in the effort of walking, so that the patient with every promenade is compelled to stop during the first few minutes. This point is of great value in diagnosis and must be closely observed (Rosenbach). Curschmann reported 4 cases of orificial stenosis of the coronaries where sudden death occurred during the act of coitus. In many cases there is no sign of dyspnœa, but there is a sharp pain under the sternum or extending to the left arm at the beginning of every bodily effort, which compels the patient to stand still or to stop his work. This condition represents the temporary occlusion of the vessels, the "claudication intermittente" of Bernard, in which the circulation adjusts itself with arrest of effort. Later on attacks of true angina pectoris may set in.

Sclerosis of the pulmonary artery is found, especially in connection with mitral stenosis and pulmonary tuberculosis, in conditions which produce chronic stasis in the lungs. Crudeli reports a case marked by paroxysmal dyspnœa and palpitation, and Romberg made a detailed report of a case which showed dyspnœa, vertigo, and marked cyanosis, with great hypertrophy of the right breast. "After any bodily effort the face had a lightning blue aspect." The cyanosis was attributed to admixture of venous with arterial blood under the powerful action of the enormously hypertrophied right ventricle.

Kidney.—Arterio-sclerosis produces nowhere more serious change than in the kidney, which undergoes gradual atrophy, with the characteristic signs of cirrhosis. Thus granular atrophy of the kidneys begins as a degeneration of the small arteries, and this degeneration owes its origin in turn probably to an altered (toxic) condition of the blood, perhaps directly to the elimination through the kidneys of various toxins. The symptoms are described in the study of diseases of the kidney. It is enough to say here that the incurability of this form of kidney disease is indicated by the character of the lesion.

Brain.—The symptoms of arterio-sclerosis of the vessels of the brain announce themselves first in a general way, and are appreciated according to the keenness of perception, education, and good judgment of the patient himself. The brain-worker becomes conscious that his work is produced under greater effort—that it does not reach its former high standard. When the archbishop asked Gil Blas to make note of and point out the first signs of defalcation in his sermons, he was beginning to feel the presence of arterio-sclerosis, and when he finally met the first notice with an outbreak of indignation the process was pronounced. The body-worker feels earlier fatigue. When the process is extensive in the brain all the organs show the signs of failing nutrition, and yet the general appearance may be that of health—that is, the individual may appear well, but he does not feel well. There is general disinclination to effort, especially to physical effort. If the brain vessels are but little affected, the mind may remain strong and clear, but there is usually much dejection of spirits, hypochondriasis or actual melancholia, irritability, alteration of disposition; later, as the process advances, occur weakening of the mental faculties, impairment of motion, vertigo, insomnia, transitory aphasia, mental disturbance, nocturnal delirium, delusions, and finally the general wreck which is characteristic of softening of the brain. Apoplexy and hemiplegia from rupture of miliary aneurysms may at any time vary the scene.

For the rest, the symptoms depend upon the site of the disease. Typical hyaline degeneration is seen especially in the small vessels of the brain and cord, and usually in association with degeneration of the nerve tissue, chronic myelitis, tabes, multiple sclerosis, syringo-myelia, progressive paresis, senile dementia, etc., dependent upon the changes in the vessels. Each of these diseases shows a characteristic symptomatology.

Arterio-sclerosis of the vessels of the extremities is indicated by coldness of the surface, cyanosis, and gangrene. Winiwarter observed the condition in a number of patients, usually in middle life, not particularly feeble, who had hitherto been regarded as entirely healthy; moreover, they had not been affected with diabetes, albuminuria, syphilis, or heart disease. These patients were suddenly attacked with rheumatoid pains in the lower or upper extremities, more frequently in the upper extremities. Almost without exception the cases were men, only very rarely women. These pains set in shortly after walking a short time, and ceased upon rest, or they showed themselves in spasmodic contraction of the muscles, or were finally of a more or less vague nature, with a feeling of formication and abnormal coldness or heat. The extremities were lightly swollen and had, as stated, a pale or cyanotic appearance. Serpentine or dilated veins were not observed. The arteries were palpable as hard cords, but without tortuousness or nodular thickenings, yet they pulsated but feebly. The pains continued to increase; the skin of the affected members became dry, fissured, and showed bright red to dark purple spots. In the course of several years—sometimes as many as five—gangrene set in under some common outside cause. The gangrene advanced relatively slowly, but always progressively. There would be an apparent cessation after separation (desquamation) of gangrenous parts and cicatrization of the wounds, but usually it was only a surgical operation which freed the patient of his disease, at least for some time. In this stage the artery—as, for example, the posterior or anterior tibial in the lower extremities—became pulseless, and even in the femoral the pulse was scarcely to be felt or had ceased altogether (Winiwarter). The symmetrical gangrene of Raynaud's disease is sometimes referred to this cause.

The DIAGNOSIS is sometimes easy and certain; sometimes it can be only presumptive; sometimes it is impossible. The diagnosis of the local condition is easy and certain when the change (hardness) in the arterial wall can be felt as in the radial artery or seen (tortuousness) as in the temporal artery.

This hardness of the wall of the artery represents clinically the highest degree of arterio-sclerosis, and refers at the age of youth with great certainty to disturbances in the circulatory apparatus, which are especially caused by heart disease, kidney disease (cirrhosis), certain intoxications (especially lead-poisoning), further disturbance of innervation on the part of the heart (neuroses, Basedow's disease) and of the vessels (Grawitz). In advanced age we must think of gout.

Curschmann divides the cases into three groups. To the first belong the patients in whom the arterio-sclerosis goes along with a marked dilatation of the beginning of the aorta and shows distinct subjective and objective signs; to the second class belong the cases in which there

are no or but few or trivial signs, notwithstanding the demonstrable existence of the condition; the third class embraces the cases in which the symptoms are grave, but are rather to be referred to some other affection. To this class belong especially the cases of isolated calcifications which are found with especial frequency at the orifice of the coronary arteries, and thus lead either to early attacks of angina pectoris or, after psychical or bodily strain, to sudden or entirely unexpected death. Patients of the second group often complain only of "nervous difficulties" until they are suddenly and unexpectedly carried off.

The first evidence of arterio-sclerosis in general is apparent in increase of arterial pressure. There soon occurs dyspnœa, which shows itself only under exercise, as in climbing stairs, the condition which Huchard designates as the "dyspnœa of effort." Subsequently there ensue palpitations, a light præcordial anxiety, coldness of the extremities, sensations of numbness, crises of pallor, violent headaches (Oettinger). Many of these symptoms have been described by Mohamed as precursors of interstitial nephritis—by Dieulafoy, less classically, as signs of "petit Brightism."

Auscultation of the heart reveals, as stated, accentuation of the second sound with metallic timbre and diastolic retardation. Sometimes an atheroma of the ascending aorta may be recognized comparatively early by dulness under the manubrium sterni, corresponding to the seat of the dilated aorta, distinct pulsation in the jugulum, and the metallic second tone at the point of auscultation of the aorta, due to increased resonance of the dilated and anatomically altered aorta.

In a large percentage of cases of affection of the aorta the pulse is altered. The change of most significance is the pulsus differens. This pulse does not depend upon peripheric changes, but upon sclerotic processes in the neighborhood of the orifices of the great vessels which issue from the arch of the aorta. This form of pulse is one of the most important signs of this affection. Curschmann found the pulsus differens 8 times in 19 cases of sclerosis of the aorta, several times after implication of only one artery—for instance, the left subclavian—occasionally with absence or a marked reduction of the pulse in the domain of two or three large vessels.

Litten found that moderate compression of a medium sized artery regularly and without exception (unless the heart's action is too much reduced) develops a peculiar spurt which gives the impression as if the blood was projected immediately under the pressure toward the ends of the fingers. This phenomenon is absolutely constant, and may be felt in all arteries not under the diameter of the radial artery, thus especially under the aorta and femoral artery. If one examines with the finger-tips of both hands, and makes pressure with the upper hand in degree which experience soon teaches, this peculiar frémissement or spurting feeling is perceived in the other hand in a remarkably distinct way. The straighter the artery and the more compressible on its substratum the more intense is the feeling. It is most easily remarked in the femoral artery and in the aorta, when the aorta can be isolated and palpated—that is, if there is not too great contraction of the abdominal muscles and too much fat. Further, it may be felt in the iliac artery, in the subclavian above and below the clavicle, in the carotid,

axillary, brachial, and finally in the radial. The aorta shows it most beautifully and most pronouncedly when it can be compressed against the spinal column; thus best in cases of enteroptosis, but the pressure must be, of course, much stronger than in all the other arteries. Here the patient is to lie, the hands are to be above and below the umbilicus, the fingers of the left hand make the pressure, while those of the right hand rest lightly upon the aorta. The spurting sensation thus felt is pathognomonic of arterio-sclerosis.

Duclos considers the radial recurrent pulse a very early and important sign of arterio-sclerosis. When it is compressed, so as to interrupt the circulation, a return pressure is felt in the artery below the point of pressure synchronous with that above it. This pulsation is attributed to the shock transmitted from the palmar arch. It may be appreciated, of course, only in cases in which the heart beat is strong. As the heart undergoes hypertrophy almost as soon as the process begins in the vessels, this sign is present at a very early period of the disease.

It is a point of differential diagnosis not unimportant that atheroma of the coronary arteries brings down the nutrition of the whole body and leads, as a rule, to a premature marasmus. This circumstance is therefore of value in considering the question of an angina pectoris which occurs in arthritic, plethoric individuals without disease of the coronary arteries, and is to be regarded either as a reflex pseudo-stenocardia—for instance, from the stomach—or as a uratic inflammatory irritation of the cardiac plexus or of the heart ganglia (Neusser). When the coronary arteries are badly affected the heart muscle suffers anæmic necrosis, with, as relics, myocarditic cicatrices, minute aneurysms, etc., the evidence of which may be shown in attacks of real stenocardia, syncope, unconsciousness, and even sudden death.

The diagnosis of chronic aortitis rests upon the age of the patient, usually past maturity, or, in the exceptional case of adolescence, the history of diseases or conditions which will have precipitated age—*i. e.* syphilis, alcoholism, hard work, saturnism, gout; upon the attacks of dyspnœa which supervene upon effort in absence of any discoverable lesion of the heart.

Arterio-sclerosis of the thoracic aorta is characterized chiefly by the peculiar difficulty of breathing, the angina pectoris, the radiating pains, chiefly to the left, but sometimes to the right shoulder—a point upon which French authors lay stress—a dulness to the right of the sternum dependent upon dilatation and elongation of the aorta.

Affection of the peripheral arteries may be revealed by sensations of paræsthesia, formication, numbness, cramps. Affection of the brain arteries runs a course partly under the picture of hemorrhage and partly under that of thrombus. On the part of the viscera there may be infarction of the kidney, of the spleen, or of the intestine.

The arterio-sclerotic shrunken kidney may not always be recognized with certainty. An abundant clear urine with a low specific gravity, small amount of albumin, and the persistence of these symptoms even after evidence of stasis has set in, may justify the diagnosis of sclerosis of the kidney vessels, and all the more if there should be hypertrophy of the heart or evidence of the arterio-sclerotic process elsewhere.

Gangrene, senile or diabetic, may be the evidence of obliteration of arterial trunks.

The individual affected in marked degree is thin, pale, sallow, shows a pronounced arcus senilis, a cranium devoid of hair, and hard, sinuous arteries. The legs often exhibit varices or phlebo-scleroses, evidence of chronic rheumatism. This coincidence is by no means absolute, but is more frequent than is commonly believed. But it must be constantly borne in mind that internal, local scleroses may show no outside sign.

In certain cases the diagnosis may be reached only by exclusion, as a gradually failing nutrition in the absence of any discoverable disease, carcinoma, tuberculosis, Addison's disease, etc., leaves no other conclusion. The age and sex of the individual should always be taken into account. The existence of previous or coincident disease, syphilis, gout, diabetes, the history of alcoholism, high living, or hard work, may point to the character of the affection.

A local condition may show itself by positive signs. Thus, attacks of true angina pectoris may indicate the existence of sclerosis of the coronary arteries; premature or general ossification of the cartilages of the ribs, sclerosis of the intercostal arteries; increase in the quantity of urine, a scanty albuminuria, uræmic symptoms, may fix the disease in the kidneys; changes in the disposition, impairment of the mental faculties, pareses or paralyses, point to affection of the vessels of the brain. Then it may be remembered that alcohol has special predilection for the liver, gout for the vessels of the kidneys, saturnism for the vessels of the kidney and intestine, syphilis for the vessels of the brain.

When there is no demonstrable change in the surface vessels and no evidence of the impairment of the action of organs, the condition of the vessels cannot be established during life.

The diagnosis of the syphilitic process rests upon the youth of the patient, the luetic antecedents, or the simultaneous evidence of syphilitic deposit elsewhere—in the skin, bones, eyes, nervous tissue, etc.

The Röntgen rays may possibly discover an early deposit. It is said that Hoppe-Seyler succeeded in making two skiagraphs by means of the *x*-rays, showing sclerotic change in certain vessels.[1] Knoll of New York got a clear skiagraph of the calcified brachial artery in a man eighty years of age.[2]

Prognosis.—The prognosis varies according to the seat and extent of the change. It depends, too, in considerable degree on the mode of life. So long as the chief organs of the body, the brain, the heart, the kidneys, show no sign of disease the prognosis is not unfavorable. Individuals may live a long life, even a life of activity, under quite extensive degenerations. So soon as hypertrophy of the left ventricle occurs the patient is put in the same position as if affected with valve disease —for instance, stenosis of the aorta. But with good nutrition the heart muscle can grow and be permanently endowed to furnish additional force. Under these circumstances hypertrophy develops as a compensation, and this hypertrophy accounts for the enlargement of the heart at maturity and in the beginning of old age. Subsequent disturbance now depends upon the condition of the heart. So long as it

[1] *Münch. med. Woch.*, April, 1896.
[2] *New York Med. Record*, Feb. 6, 1897.

keeps in good condition, is well nourished, and by its increasing size overcomes the obstacle, there is nearly perfect health.

It must be remembered that certain alterations of the vessels may be compensatory. Thus the proliferation of connective tissue which develops in the intima and adventitia in the processes of arterio-sclerosis may be substituted and make firm lesions of structure; further, the hypertrophy of the muscular tissue, which leads to thickening of the media in the smaller arteries, assists the circulation in overcoming the obstacle offered by chronic nephritis. On the other hand, an isolated affection in the wall of a small artery may contract its lumen, or occlusion by a blood clot may immediately destroy an important centre, as that of speech or sight, or may lead to acute or chronic necrosis in the brain or cord, with paralysis, while a simple dépôt in the muscle of the heart may be the prime cause of final rupture of the heart (Bäumler).

Persistent failure of nutrition in spite of all effort at relief makes the prognosis grave. The melancholia of failing nutrition and atrophy is a bad sign. So soon as individual organs become affected the prognosis becomes grave. Sclerosis of the brain, of the heart, of the kidneys, are irremediable affections. Arrest of the process should be the most that could be expected. It is needless to say that true stenocardia, cirrhosis of the kidney, cerebral softening, have an unfavorable prognosis. The prognosis of the syphilitic process is not so good as might be imagined, as the condition is often recognized too late in the history of the case. Destructive changes will have already taken place.

The prophylaxis calls for moderation in eating and drinking from youth up, the avoidance of severe muscular strain, including the sports of boat-rowing, ball-playing, bicycle-riding, the abjuration of bad habits, the treatment of obesity, diabetes, lead-poisoning, and gout.

The prophylaxis of arterio-sclerosis requires, in the upper classes, self-denial of indulgence in alcohol and luxurious meals, and in the lower classes the ability to avoid physical strain, which leads to sudden increase of blood pressure and distention and rupture of degenerated tissue. The prophylaxis of the syphilitic process depends upon the careful and long continued treatment of syphilis, the avoidance of excitants, alcohol, tobacco, luxury, and strain.

Treatment.—The treatment must have special reference to the mode of life, which must be made temperate in all things. Alcohol should be abandoned entirely or limited only to the lighter wines. Beer is objectionable because it distends the vessels. The tendency to obesity may be limited by a reduction in the quantity of fluids, by the Oertel régime, to 34–36 ounces per day, avoidance of starchy foods, and prescription of exercise. By the use of the thyroid extract alone the writer reduced the weight of a helplessly obese woman from 386 to 286 pounds in the short space of seven weeks. The individual must, if possible, avoid brain-worry and excitement. How dangerous excitement and bodily effort may be is learned from the report of 5 cases of sudden death during coitus (Curschmann). It is not to be doubted that disposition plays an important rôle, and there are, as stated, distinct hereditary or family susceptibilities. These susceptibilities depend partly upon the real weakness of certain tissues, but especially upon the increased excitability of the nervous apparatus, whereby a relatively

trifling irritation leads to great activity and so to premature senescence. The heart wears itself out by superfluous, and for the most part purposeless, work. It is important to recognize the condition early, as it lightens the diagnosis in doubtful cases and renders it possible to delay the deleterious circumstances in the mode of life in good time (Rosenbach). The patient must himself cultivate cheerfulness, contentment, and self-control. Injunctions in this direction belong more to the domain of hygiene than therapy. They may be epitomized in the statement which Horace made: "Having dined in a temperate manner, just enough to hinder my having an empty stomach, during the rest of the day I trifled in my own house."

The question of food may not be so lightly dismissed. The doubt about the diet is shown in the discrepancy of opinions. Reference has been made already to the view of Alanus pointing to a precipitation of lime salts under a vegetable diet (p. 536). It will be remembered, however, that this view, if it should be confirmed, has reference only to the process of calcification. Arterio-sclerosis is another matter.

It is clear that the arthritic or uratic, gouty, diathesis plays an etiological rôle in the development of arterio-sclerosis. The almost constant leucocytosis, which is the expression of excessive nutrition in the arthritic diathesis, may lead, by retardation of the blood current in consequence of the *plethora ad vasa*, to the migration of leucocytes into the inner wall of the arteries, and thus excite in them pathologic proliferation processes. On the other hand, it is also possible that the deposits of decomposition of meat nutrition, rich in nuclein, whether uric acid or xanthin bases or poisonous ptomaïnes, may, on account of defective oxygenation, irritate the vessel walls under increase of pressure, and by long contact anatomically injure them, as other poisons—for instance, lead, ergotin, etc.—are known to do. From the circumstance that the atheromatous process is rare in herbivorous animals may be deduced a therapeutic hint, both in the prevention of arterio-sclerosis in disposed individuals from the standpoint of prophylaxis, and in the treatment of a developed condition with its symptoms—for instance, angina pectoris. The hint is toward vegetarianism (Neusser).—anyhow, to less food.

A life of ease and indolence certainly favors the over-distention of the vessels, and thus directly contributes to the spread of the disease. Any tendency to sluggishness of circulation, especially in the domain of the portal vein, must be counteracted by exercise, laxatives, especially calomel and Carlsbad salts, etc. The proper amount of rest and sleep is necessary, but excessive sleep favors degenerative change. Thus, the after-dinner nap is not to be recommended in persons affected with arterio-sclerosis, but the individual may retire early that he may arise early. The prevention of arterio-sclerosis resolves itself really into the study of the means of prolonging life. Men who, like Goethe and von Humboldt, still possessed in advanced age the spring of youth could have been but little affected by arterio-sclerosis.

The treatment of affection of the brain, heart, kidneys, etc. belongs to the articles on diseases of these organs respectively. Though the affections of these organs are known by their consequences in softening of the brain, angina pectoris, cirrhosis of the kidney, the arterio-sclerotic process is itself the real disease.

The only agents which deserve the name of remedies are the iodides, which are often said to be the "medicines of the arteries." The influence of the iodides in conditions which depend upon syphilis may be understood. It is more difficult to comprehend the actions of the iodides when the process depends upon other affections. It is sometimes assumed that the iodides address or neutralize some toxic principle in the blood. This subject remains as yet, however, wholly in the realm of speculation. Nevertheless, there is universal testimony as to the efficacy of the iodides in arterio-sclerosis. Huchard is especially emphatic in their use, and Benedikt insists upon "the extraordinary effect of this remedy in atheromatosis, which process it hinders." The potassium iodide may be given in doses varying from five to twenty grains, well diluted, best in half a glass of milk, before meals. When the action of the heart is weak the potassium may be substituted by the sodium salt, which is somewhat less depressing. The very best preparation under these circumstances is the tincture of iodine, in the use of which the iodine selects its own basis, and thus in no wise irritates the stomach or degrades the body. The tincture of iodine is best given in the dose of ten drops in a wineglassful of sweetened water before each meal.

It is necessary to state that much patience must be exercised and much time must lapse before any real effects may be seen, and in no cases may the iodine or the iodides be pushed to the injury of digestion. Sometimes this evil may be avoided by the observation for a time of the milk diet and the administration at the same time of arsenic in the form of Fowler's solution, of which two or three drops may be given immediately after each meal.

In the graver forms of arterio-sclerosis marked by hardness and tortuousness of the arteries, by deposits of chalk plates, and by diffuse calcification, further by hypertrophy of the left ventricle, metallic second aortic sound, and hard strong pulse, the question of treatment must have reference to the reduction of resistance in the arterial system and the prevention of blood pressure in the arteries. This indication may be met, in the presence of threatening apoplexy or even when it has already occurred, by venesection or in milder cases by diuretics and laxatives, especially as accomplished by the use of alkaline mineral waters, Carlsbad salts. Warm salt baths have a certain and, different from most other baths, safe depletory effect in cases of this kind (Curschmann).

It is often wise to support the heart, especially with strychnine or the tincture of nux vomica. In the presence of violent attacks which disturb the night's rest the best effects are obtained by small doses of morphine, $\frac{1}{6}$ grain (0.01), as in the severest attacks, even in the presence of œdema of the lungs, morphine is attended with only good results. The morphine may be combined with advantage with atropine, gr. $\frac{1}{150}$ to $\frac{1}{100}$. Sometimes inhalations of amyl nitrite, of ether, or of chloroform are of value. Nitro-glycerin may be of great value. Huchard especially recommends it in the relief of dyspnœa, headache, vertigo, etc. at the beginning of the affection. Amyl nitrite is best preserved in glass capsules enveloped in cotton or silk, which becomes saturated with the breaking of the capsule between the hands, so that the patients may inhale the drug from the apposed palmar surfaces held over the nose and mouth. A convenient combination of amyl nitrite, gr. $\frac{1}{4}$, with nitro-

glycerin, gr. $\frac{1}{100}$, menthol, gr. $\frac{1}{50}$, capsicum, gr. $\frac{1}{100}$, is put up at the Westminster Hospital in the form of a chocolate tablet which may be administered three or four times a day (Sansom). Digitalis is indicated only where there is a marked dilatation of the heart, where the heart has suffered in its nutrition, or where the pulse is quick and weak. Severe palpitation may be relieved by the application of an ice bag or the so-called heart vessel filled with ice, while the severest attacks call for the analeptics—champagne, camphor, ether, musk. The most powerful cardiac stimulant is camphor, which may be dissolved in the oil of sweet almonds in the proportion of 1 : 10, or even 1 : 4, and of this solution several syringefuls injected under the skin will at any time bridge a patient over an impending collapse. The subcutaneous injection of caffeine, the natro-benzoate, is a quick stimulant to a failing heart, but morphine, as a rule, under proper precaution, and especially atropine, are the best of all the analeptics in these cases.

Some of the so-called "animal extracts" may be utilized in the course of time. Shrinkage of calibre, with reduced nutrition from defective blood supply to the tissues and organs, "is the evil to which the artery seems to be most prone." The influence of myxœdema in contracting the vessels would indicate the existence in the blood of some chemical cause, and this fact finds additional support in the remarkable effects of the intravenous injection of the extract of the suprarenal capsules. The effect of the suprarenal body is therefore exactly the opposite of that of the thyroid gland, which dilates the vessels as much as the suprarenal bodies contract them. It was observed by Schäfer and Oliver that the injection of three grains of the gland raised the blood pressure two or three times as high as it was before. So powerful is the action of the extract upon the muscular tissue of the vessels as to almost entirely obliterate the calibre of the tubes, and the blood pressure is suddenly raised and is sustained at a height as great as that which is produced by strychnine. As the peculiar properties of the extract are not destroyed by boiling, the active principle is supposed to be an alkaloid. It is known that this principle may be extracted by alcohol.

Warm (woollen) under-clothing should be worn constantly to avoid the effect of the spastic contractions of cold.

The pain of aortitis is relieved by the ice bag, which should be suspended over the chest. Sansom especially recommends an ointment of tincture of belladonna, tincture of aconite, oil of peppermint, of equal parts, all mixed intimately with an ointment of the oil of benzoated lard. This mixture makes a creamy ointment which may be gently rubbed into the painful area by a pad of cotton wool. Severe pain calls for the subcutaneous use of morphine.

Tepid or warm baths of long duration have a very favorable effect in the early stages. Baths are of great value in the treatment of plethoric states. The Hot Springs of Arkansas and Virginia furnish here the necessary requirements, with pure atmosphere and relaxation from domestic and business cares. Baths hot and cold may be used at home, and diaphoresis may be secured by enveloping the patient in a blanket after a hot bath. These baths may be made more stimulating by the addition of one pound each of common salt and sea salt.

Baths have usually been considered contraindicated in arterio-scle-

rosis on account of the danger of contraction of the cutaneous vessels, increase of pressure, and rupture of miliary aneurysms in the brain. With baths containing salt, and more especially carbonic-acid baths, the contraction of the vessel gives way very rapidly. The danger of increase of pressure exists, therefore, only in the beginning of the bath, and is due largely to the sudden change in temperature. This evil may be avoided by having the patient gradually exposed. The surface may be moistened at first, and the patient let into the bath very gradually, as in a half bath, while the upper part of the body is protected and the head is covered with cold compresses to prevent congestion of the cerebral vessels. The bath should have a temperature of at least 31° C. (88° F.). With these precautions baths have no bad effects in arterio-sclerosis. On the contrary, by increasing activity of the heart and relieving heart weakness they are very beneficial. But baths should not be taken for six months or a year after an apoplexy, embolus, or thrombosis of peripheral vessels.

Massage is often of value in emptying distended bloodvessels and securing more equable distribution of the blood. The actual efficacy of massage was demonstrated by Eccles, who found with instruments of precision that the quantity of blood distributed through the tissues was directly facilitated, and in this way the work of the heart was lessened. Moreover, the blood is retained longer in the tissues under the influence of massage.

Failing digestion may be assisted with dilute hydrochloric acid and bitter tonics, cinchona, cascara, nux vomica, and failing nutrition supported by cod-liver oil, malt extract, etc.

Dropsical states, especially in connection with chronic nephritis, may be relieved by diuretin, gr. xv, in half a glass of Seltzer water every four hours, or by calomel (which is, however, not quite so much indicated in kidney disease), 3 grains three times a day for two or three days, or by toning the heart with digitalis in the form of a tincture, 10 drops every four hours. The calomel may be combined with digitalis, each 1 grain in powder with 5 grains of sugar. The precaution must be taken with the using of calomel to brush the teeth after each meal, to cleanse the mouth with borax, or take internally of a saturated solution of potassium chlorate and peppermint water a teaspoonful every two hours. Obstinate cases call for the use of puncture, especially by the insertion of tubes under thorough asepsis.

In the beginning of gangrene the extremities should be treated with rest and elevation, with strengthening of the heart, and with warm baths. Massage is to be avoided; on the other hand, an attempt may be made with the usual doses of potassium iodide, or, preferably, with the tincture of iodine.

Even under manifest gangrene a spontaneous cure may occur, though in fact it happens but very seldom; but in the absence of threatening symptoms one should postpone operation, as usually the disease is not ended with *one* operation. The operation should still the pains. The line of amputation should lie in well nourished tissue and should heal. Any general rule for the place of amputation may not be given, but of course one must act more cautiously in the case of the hand than of the foot (Winiwarter).

The treatment of the syphilitic process calls especially for the energetic use of mercury in the form of the ointment and the internal use of potassium iodide. Pains are especially relieved by the iodide salts.

Patients whose means afford it may prolong life by avoiding the rigors of rough weather in change of climate.

The same means which tone the muscle of the heart will also strengthen the muscle wall of the vessels. Experience has shown that graded exercise in the open air is the best tonic to all muscle structure, unstriped and striped. So choice may be had with reference both to prophylaxis and to treatment, and with cautious adjustment of the needs and condition of the individual case between sailing, rowing, riding, walking, moderate gymnastics, always in the open air, mountain-climbing, hunting, etc. Lord Palmerston's saying, which has done so much service elsewhere, may be cited here: "The outside of a horse is the best thing for the inside of a man." The bicycle is an economical substitute of nearly equal value.

In seeking the proper climate it must be remembered that moderate elevations, 1500 metres, may be well tolerated, but that great elevations throw increased work upon the heart. The occurrence of dyspnœa, nervousness, and especially insomnia, indicates the necessity of change to a lower level.

The bloodvessels are toned, as stated, in the same way as the heart, by gentle exercise systematically graded, especially by ascending gentle acclivities, with here and there a rest in shady spots. The lower ranges of Maryland, Virginia, and the Carolinas furnish the proper elevations, with combinations of sunshine and shade, and at various places the refreshing influences of baths, hot springs, warm springs, healing springs, sulphur springs, etc., in the treatment of chronic changes in the bloodvessels. To avoid the harshness and vicissitudes of fall and winter the patient should migrate with the birds to the South, where he may find the best climate in Florida, Georgia, and Alabama. The Bermudas, the Bahamas (Nassau), which are now next door, furnish a more relaxing climate, while somewhere in California may be found every variety of mildly stimulating and invigorating season.

Out-door life favors oxygenation of the blood—favors metabolism and rejuvenation of all the tissues.

ANEURYSM.

Definition.—Aneurysm (*ανεύρυσμα*, a widening), arterio-ectasia, is a local—*i. e.* more or less circumscribed—dilatation of an artery. The dilatation may involve all the coats or only one or two, and may be in shape sacciform, fusiform, or cylindrical. The sacciform is the most frequent. The form of the sac may be spherical, oval, elongated; the sac may be, further, bottle-shaped, lobulated, sessile, or pedunculate. The orifice of communication with the artery may be large or small; at first it is small and irregular.

History.—Galen, Ætius, Avicenna, derived aneurysm from rup-

ture of and injury to an artery. Vesalius found it caused first by distention of the artery wall, later by rupture. Fernelius ascribed it to stretching of the coats of the wall of the artery. Hildanus thought that an aneurysm was caused by a rupture of the internal layers; Pelletan believed that the rupture of the intima gave rise to the formation of an aneurysm. On the other hand, Monroe considered only those tumors aneurysms in which all the membranes of the wall of the artery were dilated. Based upon numerous investigations, Scarpa maintained that a dilatation was an aneurysm only in cases marked by rupture or disease of the intima and dilatation of the other layers. The intima is first roughened, calcified, and ulcerated; the media may be later eroded, then the adventitia, while a newly formed connective tissue constitutes the wall of an aneurysmal sac. Hodgson and Burns first subscribed to the view of Scarpa, but held later that there are aneurysms in which all the walls of the artery are affected, and explained the absence of certain layers as due to rupture from distention. Kreysig considered the chief cause of aneurysm to be inflammation of the arteries, which led to erosion, ulceration, and steatomatous degeneration. Guthrie maintained that originally the media and intima were uninjured, and were affected only with the increase of the aneurysm. Astley Cooper, Sabatier, Dupuytren, and Richerand advocated the dilatation of all the coats of the arteries with rupture of individual layers.

Bizot distinguished two affections of the wall of the artery—one attended by the formation of cartilaginous plates and inflammation, and the other an atheroma with degeneration of the membranes without inflammation. Rokitansky also subscribed to this view. Lobstein separated ectasia from aneurysm (Neudörfer).

In ancient times attempt was made to reduce the blood pressure by frequent venesection. This treatment was especially practised by Albertini, though it is usually associated with the name of Valsalva. It consisted in the frequent repetition of bloodletting at certain intervals, with rest and inanition.

Velpeau (1826) was led to adopt acupuncture, with the observation that the introduction of a needle into the femoral artery of a dog led to a deposit of fibrin and the obliteration of the vessel in less than four days. Home (1796) had long before introduced heated needles into an aneurysm of the iliac artery. This method got a new impulse in the method of Pétrequin (1845), who introduced the galvanic current into the sac with a needle as an electro- or galvano-puncture. Duncan and Frazer made many experiments to establish the effect of galvanopuncture, while Cinicelli actually put the method in practice in the treatment of aneurysm. John Hunter (1790) made the most important contribution ever made to the treatment of aneurysm in distal ligation of the artery. Miliary aneurysms were demonstrated by Charcot and Bouchard in 1872. Varicas injected tannin in 1845; Malgaigne injected the perchloride of iron in 1853; and Valette and Pravaz injected perchloride of iron (méthode Pravaz, Paris, 1857). Tufnell wrote on the treatment of aneurysm by compression in 1849, and by position in 1873, reporting the successful treatment of internal aneurysm by rest and restricted diet, illustrated by cases in hospital and private practice (Lon-

don, 1864). Macewen presented his method of needling an aneurysm, to induce the formation of white thrombi within the sac (1890).

The new points in etiology demonstrate the overshadowing influence of syphilis in the production of aneurysm. Potassium iodide in treatment was first recommended by Bouillaud (1858), and later by Chuckerbutty in Calcutta (1862), but found its warmest advocate in Balfour (1868).

Aneurysms were divided in antiquity into two groups—true and false. The false depended upon injury of the artery.

General Considerations.—Dilatation may occur in the course of any artery in the body. Hence aneurysms may vary in size from the microscopic or barely visible, so-called miliary aneurysms of the brain to the great tumors developed upon the aorta, which take up a large part of the cavity of the thorax and abdomen. Phänomenow reported a case of congenital aneurysm of the abdominal aorta, which on account of its magnitude proved an obstacle to delivery.

Aneurysm is in general a rare affection. Excluding miliary aneurysms, which have a different genesis, among a million of men there may be one aneurysm. The fact is that aneurysms are seen only in the course of decades by physicians or surgeons with the most extensive practice, and this fact makes the diagnosis difficult. Aneurysm is rare in Germany, France, and Italy—is more frequent in England. The frequency in England has been accounted for by alcoholism, excessive meat diet, and gout. But muscle fibres differ in energy as well as bulk. Thus among the average subjects in America and India the muscle may be narrow and thin, but these apparently atrophic muscles may carry heavy burdens which the Europeans and the Creoles with their powerful musculature cannot sustain. The majority of the cases of aneurysm in New York occur among foreigners (Hirsch). Soldiers and civilians are affected with aneurysm in the proportion of 11 to 1 (Lawson).

According to all statistics, the great bulk of cases occur between the second and fourth decades of life. Of the 92 cases tabulated by Hayden, 60 were found in individuals between the ages of thirty and fifty. Aneurysm is more frequent in men than women. With the exception of the worm aneurysm of the horse, the condition does not occur in the lower animals.

Site of Aneurysms.—Dilatation occurs more frequently in the external than in the internal vessels, because of the greater exposure to injury and less support from circumjacent tissue. The artery most frequently affected is the popliteal, next the crural, then the carotid and axillary arteries.

Billroth found among 23,000 patients, whom he had seen in the clinics at Zürich and Vienna from 1860 to 1892, 26 cases of aneurysm of the extremities and neck. Of these 26 cases, 15 were of traumatic origin and only 11 were spontaneous. Of these 11, 8 occurred upon the popliteal artery, in 1 patient on both sides, 2 on the carotid artery, 1 on the subclavian artery. Aneurysm of the great arterial trunks is, fortunately, very rare.

Litten found, of 25 cases of aneurysm of the aorta, affection of the ascending aorta and the arch of the aorta 12 times; of the left innomi-

nate, including its great vessels, 10 times; of the descending thoracic aorta twice, of the abdominal aorta but once. Of the 32 aneurysms collected by Briggs, 26 were of the thoracic aorta, 4 of the abdominal aorta. Of the thoracic aneurysms, 12 affected the ascending aorta, 8 the arch, 4 the descending aorta.

Crisp found in 551 cases the seat of aneurysm as follows. We cite only the internal aneurysms:

Thoracic aorta	175	4.1 per cent.
Abdominal aorta	59	
Pulmonary artery	2	.03 " "
Innominate artery	20	3.6 " "
Subclavian artery	23	4.1 " "

Of 109 aneurysms of the aorta, Myers found affected the—

Ascending aorta	37 times.
Arch of the aorta	38 "
Descending aorta	19 "
Abdominal aorta	15 "

The frequency of aneurysm of the aorta is somewhat greater than given, because statistics are derived wholly from post-mortem examinations, and, as is well known, certain cases pass unrecognized. The popliteal is the only artery which may at all compete with the aorta in the frequency of affection. Aneurysm of the aorta and of the popliteal artery includes two thirds of all the cases. External aneurysms belong to the domain of surgery.

Internal medicine is concerned chiefly with the consideration of aneurysm of the aorta and its main divisions in the chest and abdomen, and the more minute aneurysms which develop in the arteries of the internal organs, especially of the brain. Aneurysm affects the internal cerebral arteries in the order of frequency as follows: middle cerebral artery, 44; basilar, 41; internal carotid, 23; anterior cerebral, 14; posterior communicating, 8; anterior communicating, 8; vertebral, 7; posterior vertebral, 6; inferior cerebellar, 3—total, 154.

Sub-varieties.—"Erosion aneurysm" is typically represented in affections of the arteries in consequence of ulcer of the stomach or in gangrenous processes. Aneurysm of the small vessels is most frequent in tuberculosis, and rupture of them is the most common cause of fatal hemorrhage. The so-called "bone aneurysm" is not an aneurysm at all, as there is no afferent or efferent artery and no aneurysmal sac. "Bone aneurysms" are really hemorrhagic sarcomata.

Where the orifice of rupture is small and the blood oozes gradually into the surrounding connective tissue, it constitutes what is called a "diffuse" aneurysm. This form of aneurysm is most often encountered in the arch of the aorta and at the abdominal aorta, where the inflammation (cellulitis) may be very painful. When the blood is discharged between the internal and middle coats it may separate these coats and burrow its way between them, or may open externally at some distance from the internal orifice. Such a condition is known as a "dissecting" aneurysm. Sansom reports the case of a soldier who had suffered from aneurysm of the aorta for more than twenty-nine years. On autopsy it

was found that a dissecting aneurysm had encircled the aorta in such a way as to form a double tube around it in its whole length.

Curious are the cases of dissecting aneurysm in which a new communication is made with the bloodvessel lower down. Bostroem reports such a case in which an aneurysm was cured in this way. Of 27 cases reported by Biggs in which there was only one aneurysm, in 21 it was saccular; in 4, spindle shaped; 2 were cases of dissecting aneurysm.

FIG. 26.

Dissecting aneurysm on a cerebral vessel, simulating miliary aneurysms (Schmaus).

Number.—As the process of arterio-sclerosis is often diffuse, it is not surprising to learn that aneurysm is sometimes multiple. Thus MacKellar reported the case of a man, aged thirty, in whom the right femoral artery had been ligated for popliteal aneurysm. Five months later the left femoral was ligated for the same condition on the left leg, and four years later the right common carotid and subclavian arteries were ligated in relief of aneurysm of the innominate, which ruptured, nevertheless, in three months after the operation. Hulke reported the case of a man affected with aneurysm of the aorta, of both external iliacs, of both femoral and of both popliteal arteries. In 2 of 32 cases of Biggs there were multiple aneurysms. Jona reported the case of a man aged thirty-three who suffered from syphilis for nineteen years, and who succumbed to a valve lesion of aortic insufficiency. The aorta was thickened at its origin, the intima showed a few calcareous deposits and ulcers, the valves were shrunken and thickened, but the most remarkable condition was the existence of nine aneurysms of the aortic wall 4 cm. below the valves, varying in size from a pea to a hazelnut.

On the other hand, a single aneurysm is sometimes found in an aorta which is everywhere else perfectly healthy. It was the observation of such a case that led Sutton to say: "I have had a sense of awe on looking into the body and seeing that while all the other organs and tissues were so exceedingly healthy, death had been caused by so limited a disease."

PATHOGENY OF ANEURYSMS.—Aneurysms arise from outside cause (trauma), inside cause (embolus), or intrinsic cause (arterio-sclerosis). Trauma belongs to surgery; embolus will be discussed later (p. 589).

Whether a degeneration shall produce the changes considered under the article on Arterio-sclerosis (p. 525) or shall eventuate in aneurysm is more a matter of degree of degeneration in a certain region than a difference of kind.

A localized process which involves extensively or actually destroys any one coat of an artery is liable to produce an aneurysm. Sometimes the intima is split or divided, while the force of the media is weakened. The blood under its constant pressure may then insinuate itself under the intima and dissect up a large part of its tract, or the pressure may by usury consume much or all of the middle coat and bulge the outer

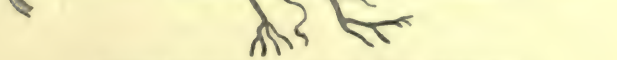
coat into an aneurysm. It is especially the middle coat which suffers in the first formation of an aneurysm. The muscular tissue is thinned, and may eventually entirely disappear. In a case of diffuse dilatation of the ascending aorta in a woman æt. seventy-two Puppe found, besides the well-known changes in the intima and media, a remarkable atrophy of the elastic elements, which under a high magnification by means of the Manchot method appeared as a network of fibres as thin as a cobweb. Köster and Krafft derive the whole process from a mesarteritis, and ascribe the weakening and destruction of the media to inflammatory changes brought about through the vasa vasorum. Weakening of the media may be counteracted for some time by hyperplasia of connective tissue, especially in the adventitia, but eventually this hyperplasia is overcome and dilatation develops.

According to Puppe, microscopic examination shows infiltration of the vasa vasorum beginning in the adventitia and continued in the media. Here and there are to be seen spots of typical granulation tissue, others of cicatricial tissue. Elastic elements are separated by the infiltration, and are sometimes ruptured. In some cases there is total rupture through the intima and media, and the adventitia is bulged. Of particular interest was a case in which there were found giant cells in the midst of an infiltration about a vas vasorum.

An aneurysm develops as follows: There is a region of necrotic tissue in the domain of a vessel, and a sudden cessation of the continuous media at the neck of the aneurysm where the vasa vasorum are infiltrated. The media discharges its important function up to the point of the necrotic tissue, where it has lost its extensibility and contractility on account of the infiltration of the nutrient vessels. When the necrotic process extends farther this most important layer of the vessel loses its power of resistance. It yields to pressure, and finally suffers rupture with the formation of aneurysm (Puppe).

Miliary Aneurysms.—Aneurysms which develop in the smaller arteries of the internal organs vary from microscopic size to that of a pin's

FIG. 27.



Miliary aneurysm on a cerebral artery (Schmaus).

head or pea. Such dilatations upon the vessels are known as miliary aneurysms. Sometimes they are but few in number, but, as a rule, they are multiple, and the vessel may look as if strewn with them. They are of variable color, but are usually dark red, and are so friable as to be easily ruptured. As a rule, they are best demonstrated by seizing the main branch of the vessel with the forceps and drawing it through water; as the smaller branches float out under this treatment, adherent particles are detached and the aneurysms become distinctly visible. Virchow described them first as ampullary ectasiæ of the vessels. These aneurysms are so adherent to the vessel wall as to appear to be

sessile growths, but Eichler as the result of his studies found them to be due to arterio-sclerosis, and thus established their nature as true aneurysms. The relation of miliary aneurysm to cerebral hemorrhage is of late much disputed (Stein).

The rapidity with which an aneurysm may develop will depend, aside from the strength of the sides of the vessel, upon the blood pressure; that is, largely upon the force of the heart; so that the view that an aneurysm has traumatic origin is, in a sense, correct. Aneurysm of the aorta certainly grows more frequent as we approach the heart. Thus, aneurysm is, as stated, most common in the ascending aorta, next at the arch of the aorta, next in its largest branches, the innominate and subclavian arteries, then in the descending thoracic aorta, abdominal aorta, and its branches. (See Plate IV.)

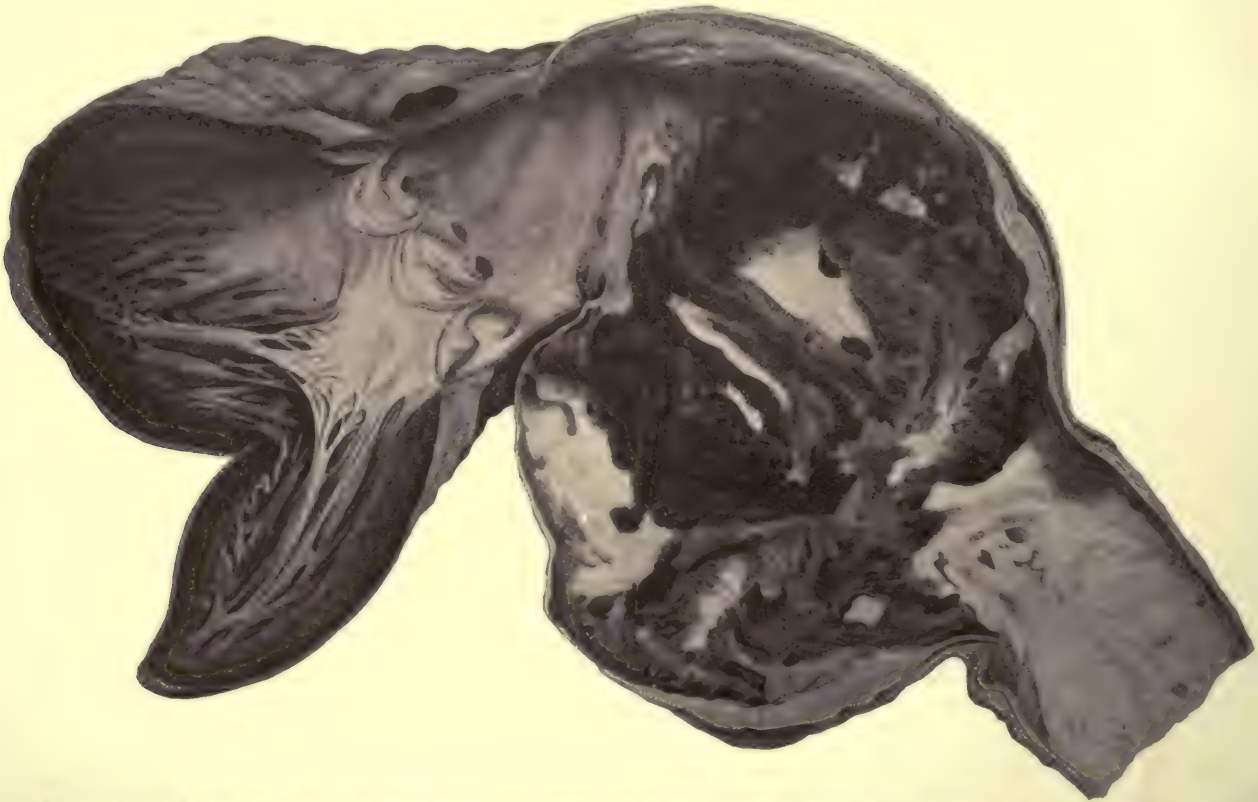
Contents.—Aneurysms are filled with blood. In most cases the contents of an aneurysm are fluid, and the pulsations of the heart may be felt in it as one of the distinguishing signs of the condition. But in many cases the blood coagulates in layers. This coagulation occurs more especially in aneurysms of the sacciform character, where the orifice of communication with the artery is small, and where the force of the current, as in a bayou, is much reduced. Thus, there may be deposited upon the wall of the aneurysm layer upon layer of coagulated blood. These layers may in turn become organized, and firm, tough yellow adherent thrombi strengthen the wall of the vessel as if by the addition of new coats. Sometimes the deposit is so great as to obliterate the aneurysm altogether, and this process constitutes the natural cure. Every effort of the physician and surgeon, by every possible means, is directed to secure this happy result. For the most part, however, the process is incomplete. The layers which line the sac are defective in places, and the distention process continues. Then, salutary as is the process of coagulation, which should be favored in every way, it is, nevertheless, not devoid of danger in that particles may be washed away by the current of blood and deposited in distant places to constitute emboli, with their peculiar consequences.

Etiology.—The causes of internal aneurysm are the diseased conditions which weaken the wall of the vessel.

That mere destruction of the layers may not suffice to produce an aneurysm was proved long ago in the experiments of Hunter and Home, who exposed the carotid artery in a dog and dissected off layer by layer, until finally the blood could be seen through the last layer. The dog was killed in three weeks. The wound had healed entirely, and the artery had neither increased nor diminished in diameter.

Quincke also tried to produce aneurysm by injury of the inner surface of arteries, but the aneurysms produced in this way were small and rapidly filled with clots. Thus an aneurysm fourteen days old had become reduced to the size of a pea and the opening was filled with a soft clot. According to Crisp, Amusat, Jones, and others tried to produce aneurysms experimentally by rupture of the inner and fibrous layer without effect. Finally, Home exposed the femoral artery of a dog—filled the wound with lint to prevent closure and to remove support from the artery. Home killed the animal in six weeks and injected

PLATE IV.



Aneurysm of the Arch of the Aorta (including part of the Ascending and Descending Aorta), Lined with Yellowish Layers of Fibrin. The Rest of the Aorta shows Atheroma. The Tumor, which had Reached the Size of a Child's Head, had Produced Broncho-Stenosis with Injury of Thoracic Vertebræ. (Lehmann's Atlas.)

the artery, finding it neither increased nor reduced in size. The artery was normal.

But that aneurysm is not produced simply by atheroma is shown by the preponderance of cases in middle life, when atheroma is not so common as in old age. Neudörfer furnishes a far-fetched view when he finds that aneurysm depends upon the formation of new tissue, not upon the stretching of old elements, and upon the rhythmic pulsation of the new elements. Where one of these two conditions is absent there is no aneurysm. The rhythmic pulsation of the new tissue is accounted for by the fact that the pulsation in aneurysm is stronger than in other normal sections of the artery. The opposite condition is found, he says, in paralytic hypertrophy, in which the number of primitive fibres may be very large, but the energy may be small or reduced to *nil*. The fact is, that a number of conjoined causes must conspire to produce an aneurysm. These causes are arterio-sclerosis (usually from syphilis), a certain age, and physical strain. The infrequency of the conjunction of these causes accounts for the rarity of aneurysm.

The disease conditions which cause an aneurysm are described under Arterio-sclerosis (page ——), and require, therefore, no detailed repetition here. As the mechanical factor plays such an important rôle in the production of aneurysm, it is not surprising to learn that the majority of cases occur during the age of greatest strain—to wit, at and about maturity. Aneurysms are infrequent in youth, and are not frequent in advanced age, save as survivals from maturity. The age of hard work is the age of aneurysm. The large contingent of cases occurs among laborers, "the hewers and drawers," porters, and artisans whose work demands severe or sustaining physical effort. The greatest number of cases occur, as stated, during the third and fourth decades of life. Of the 16 cases diagnosticated in life by Puppe, 13 were men and 3 women—a relation which corresponds to that generally accepted. Of the men, 1 was a waiter, 3 were masons or allied artisans, 1 turner, 2 weavers, 2 basket-makers, 1 hunter, 4 civil officers; thus, 4 led sedentary lives, 9 were artisans. As to age, 3 were over sixty, 2 between fifty and sixty, 4 between forty and fifty, 6 between thirty and forty; and of these 5 were about thirty-five and under; no one was under thirty-four.

It is a recognized fact that aneurysm is chiefly caused by arterio-sclerosis. But it has been often objected that arterio-sclerosis occurs preferably in advanced life, while aneurysm reaches its greatest frequency at the age of forty, and that arterio-sclerosis is a very frequent and widely extended process, while aneurysm, on the other hand, is extremely rare. Now, Thoma has shown in the most convincing way how this apparent contradiction is to be explained. A dilatation aneurysm arises under temporary variations of arterial pressure, when the wall of the artery suffers from imperfect and diminished elasticity. These conditions are fulfilled when in the beginning stages of diffuse arterio-sclerosis the tunica media is weakened and is diffusely distended, but the intima is not yet sufficiently strengthened by connective tissue new formation. As now the beginning stages of diffuse arterio-sclerosis occur at the age of thirty-five to forty, while at the same time the male population at this age is capable of quicker and more powerful muscular

effort, whereby there is increase of blood pressure and thus a possibility of the formation of dilatation aneurysm, at no other time of life are the conditions for the occurrence of an aneurysm more favorable than just about the age of forty. The great rarity of aneurysm in comparison with the frequency of arterio-sclerosis is explained, first, by the fact that the female part of the population only rarely suffers from aneurysm, because (*a*) in them arterio-sclerosis undoubtedly sets in later; (*b*) women are seldom subjected to the forms of severe bodily strain which lead to sudden marked increase of blood pressure; secondly, because the men are subjected to the prerequisites for the development of an aneurysm only for a comparatively short time, and during this relatively short period the exciting cause is usually absent (Lubarsch).

It is a curious fact that locomotive engineers and firemen, who are subject to frequent agitations, jerks, and vibrations, are rarely affected with aneurysm, while sailors, whose avocation protects them against these vibrations, are frequently attacked.

Strain.—Aneurysms develop, as a rule, so insidiously as to escape notice until they have reached a certain size, but sometimes the period of inception is definitely known. These are the cases in which the vessel is dilated under the influence of a sudden strain, premising, of course, a diseased condition of the vessel in all cases. Where the vessel walls become weak from any cause it may be understood how a sudden increase of blood pressure, as after lifting a heavy burden, wrestling, or rowing, or sudden fright, with arrest of the heart, may produce dilatation. During the preparation of this article the writer presented to his class a typical case of aneurysm of the ascending aorta in a man, aged thirty-nine, who gave his avocation as a steam-fitter—that is, a joiner of steam pipes—in the execution of which work he was compelled, he said, to sustain above his head the weight of heavy iron pipes. This patient, it may be stated, had had no subsequent syphilitic symptoms, but could recall the existence of a sore upon the penis several years ago. Not at all infrequently it may be heard in the history that the patient dates his disease from some sudden or more sustained effort where every muscle is strained to the point of exhaustion. In the case of the woman whose aneurysm is shown in Fig. 29, p. 563, the tumor appeared five months after a violent wrench in the endeavor to extricate herself from the machinery of the laundry in which she had been caught. This patient declared, as in the case of the joiner cited, that she had pains, palpitation, and dyspnœa, though not constantly, from that time on. Lancisi reports the case of a runner, aged forty-five, in whom an aneurysm of the aorta followed a blow upon the left side of the back, and Litten mentions the case of a man who in falling backward partially ruptured the aorta and developed a dissecting aneurysm. The writer once saw reported the case of a physician who developed an aneurysm in consequence of almost superhuman traction upon the obstetrical forceps. Anything which raises local blood pressure may have this effect: the act of parturition, severe straining at stool, the act of coition have frequently served to start an aneurysm or at least to make the condition manifest. So, too, it may be understood how, as in the cases reported by Tufnell and Ogle and demonstrated by the

studies of Church, Ponfick, and Pel, an aneurysmal dilatation may occur above an embolus or excrescence detached from a heart valve or from an atheromatous ulcer blocking a small vessel.

Syphilis.—Syphilis is of all the causes the overshadowing factor. Late investigations show that circumscribed aneurysm the result of arteriosclerosis, in distinction from the more diffuse variety which may be produced by other causes, is almost without exception the result of syphilis antecedent eight to twenty years. Especially is this the case in patients in or between the fourth and sixth decades of life. The relation of syphilis has been recognized from the earliest times. Thus, Marcus Aurelius Severinus attributed aneurysm to the "syphilitic cachexy." Paré, Lancisi, Albertini, all mention syphilis as a cause, as did also Morgagni. Jaccoud found 22 cases of aortic aneurysm in syphilitic patients who also had syphilitic aortitis. Von Langenbeck declared that in one-half of all the cases of aneurysm of the aorta which he saw the patient had suffered for a long time with syphilis. Maclean had the same experience with soldiers, and Welch found in 35 cases among soldiers 8 probably, and 17 certainly, syphilitic. Verdié reported 32 cases of syphilitic aortic aneurysm.

The rôle of syphilis is indicated also in the statement of Aitken, who found 50 per cent. of cases of aneurysm of the aorta in soldiers affected with syphilis. Malmsten discovered syphilis in 80 per cent. of his cases, while alcoholism could be adduced as a cause in but 5 per cent. of cases. It is observed that aneurysm is much more common among courtesans than among other women.

Fränkel made a recent demonstration of heart syphilis before the Berlin Medical Society. He has repeatedly called attention to the development of aneurysm from syphilis. An English author, Welsh, had maintained that syphilis was the cause of 60 per cent. of cases of true aneurysm. Other authors go further and put the percentage as high as 80.

Of Fränkel's 19 cases, 9 were syphilitics, and none of the 9 had reached the age of fifty. The percentage of cases caused by syphilis was 47. The demonstration was upon a case which showed extensive sclerotic changes in the aorta, along with unmistakable signs of heart syphilis. The patient had made frequent complaints of headache, with occasional attacks of syncope. Rheumatism had existed in former years, and the symptoms which the patient showed of insufficiencies of the aortic valves had been derived from rheumatism. The history showed that the husband had been syphilitic. The patient, a woman, had suffered formerly with tumors of the head that had discharged and left cicatrices. She left the house improved, but returned with symptoms of heart disease, especially with violent attacks of angina pectoris.

Though the good effects usually attributed to the iodides are commonly ascribed to the influence of these agents upon syphilis, the disease process in the aorta is rather a remote than a direct consequence of syphilis. In other words, aneurysm occupies the relation to syphilis of tabes dorsalis, progressive paresis, and other scleroses which develop often decades after the original infection. Thus, Verdié and Fournier put the period of development of aneurysm at eleven to eleven and a half years, and Puppe at sixteen years, after the original infection.

The objections to the syphilitic origin of atheroma and aneurysm hitherto obtained were based upon the absence of the histological character of syphilitic tissue change, but as it is now known that in tabes, a disease which is acknowledged to be largely caused by syphilis, there is no syphilitic change in the cord, this origin of aneurysm may be accepted in the absence of giant cells in the wall of the aneurysm. When we remember that in the florid stage of syphilis multiple nodules of maculo-papular exanthem develop in the skin with small celled polyvascular infiltration and thickening of the walls of the smallest vessels, we may understand the changes that take place in the vessel walls of the larger arteries and veins; and as we see sometimes permanent changes in the skin, we may understand how the same persistence may characterize the changes in the vessels. The wall of the vessels, like the skin, may suffer circumscribed necrosis in the media, so that the elastic lamellæ become more extensile and fragile and the blood pressure may produce an aneurysmal dilatation (Bäumler). But characteristic syphilitic changes have been discovered and described. Thus, Kalindéro Babes reported 3 cases, including 1 of Herz, of syphilitic and aortic aneurysm in women of fifty and thirty-four years, and 1 in a young physician of twenty-five years, in whom a careful microscopic examination of the affected aorta was made, with findings as follows: The aneurysms were small circumscribed dilatations in consequence of gummatous disease of the vessel wall. The seat was usually the concavity of the aorta. Sometimes there were associate changes in the aorta or neighboring organs. The gumma of the aortic wall had softened and formed the point of least resistance, inclining to perforation. Sometimes the intima had been ulcerated by the gumma and was secondarily affected with pyogenic micro-organisms. The histological investigation of the artery wall showed the characteristic syphilitic sclerosis of the vasa vasorum, which had produced obliteration.

Alcohol is the next most frequent factor in the production of aneurysm, but the effects of alcohol are sufficiently dwelt upon in the discussion of Arterio-sclerosis (page 533). Duplaix declares that the number of cases of aneurysm diminished considerably in the hospitals of Dublin during the temperance crusade in Ireland, but increased again after it was over.

As the same indulgence which leads to syphilis leads also to the use and abuse of alcohol, it is not surprising to learn that these influences are often conjoined in the same individual.

The effect of *gout* and *rheumatism* in producing degeneration of the wall of arteries is sufficiently emphasized in the discussion of Arterio-sclerosis (page 535).

That *shock* or more sustained depressing mental emotions, though they may not suffice to produce an aneurysm *de novo*, will, by degrading the general nutrition, favor the weakening process in a diseased vessel, has been repeatedly remarked. Thus we may understand the statement of Lancisi when he declared that he knew of a number of cases of aneurysm which developed in consequence of excessive fear occasioned by an earthquake.

Symptoms.—*Aneurysm of the aorta* is always latent for a time, and is sometimes latent for a long time. Feilchenfeld reported a case of

aneurysm of the abdominal aorta of the size of a child's head protruding to the right of the abdomen in a woman aged eighty, who had never made any complaint whatever up to the day of rupture and death. In a case reported by Biggs there was found in a nurse who had only shortly before shown grave symptoms an enormous aneurysm of the ascending aorta and arch, 15 by 20 cm. in size. The heart was displaced downward and to the right; no rupture.

Internal aneurysm is always quite difficult of discovery at first. In fact, aneurysm of the aorta, the study of which chiefly engages the attention of the physician, may remain for a long time latent—as often as, according to Malmsten, one fifth of all the cases. Sometimes an aneurysm reveals itself only by the final catastrophe, and such cases of sudden death sometimes assume juridical importance. Thus, Litten reported the case of a man, aged forty, who fell dead during the examination. Upon autopsy it was seen that death was caused by rupture of the aorta, just above the valves, into the pericardium, which was found full of blood. Mention is made later of similar cases reported by Draper. Biggs declares rupture of an aneurysm to be one of the most frequent causes of sudden death without previous grave symptomatology. This statement is irreconcilable with the rarity of aneurysm.

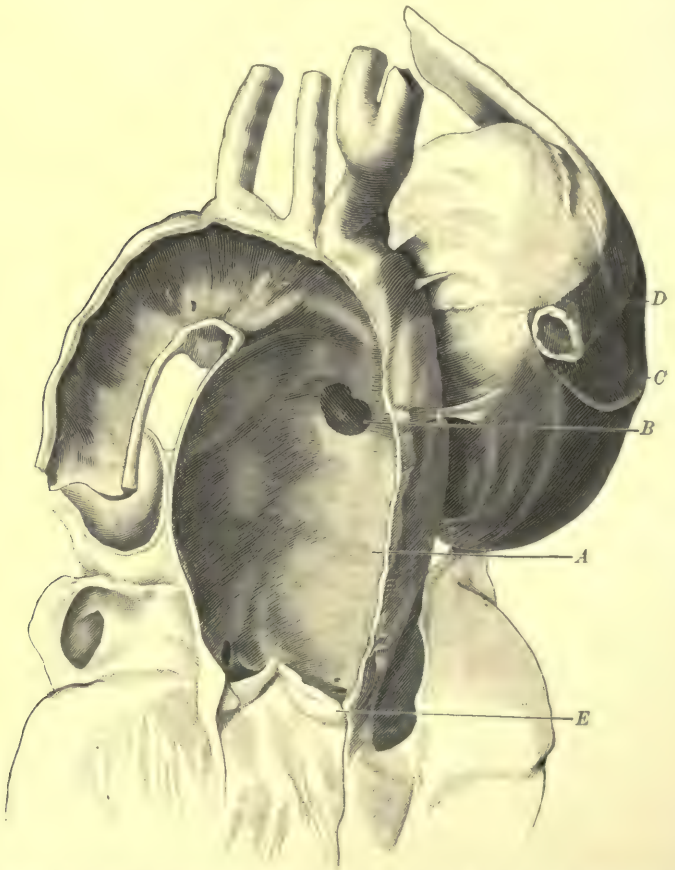
Pain.—As a rule, the first signs are vague and are misinterpreted. The most prominent, persistent, and characteristic symptom of aneurysm of the aorta is pain. The condition is often announced with an attack of pain, which may vary in every degree of intensity. It is often so light as to be mistaken for pleurodynia or intercostal neuralgia. Sometimes, on the other hand, the pain is the excruciating agony of angina pectoris. The pain is not persistent, as a rule, but comes and goes in the course of the disease, and is connected, especially at first, with effort. As a rule, the patient complains of dull pain in the chest and abdomen, sometimes with palpitation. Hereupon the pains greatly diminish, and the pressure is not so severe, because the various organs accommodate themselves to the tumor. Difficulty is then experienced only after bodily effort, heavy meals, or in certain positions of the body. In the course of time paroxysms occur at rest, and, as the aneurysm makes more decided pressure, the pain becomes more persistent, though at all times it varies especially with the force of the heart—that is, with the pressure of the blood. But pain is such an universal expression of disease as to have value in the diagnosis of aneurysm only when it is unusually persistent or severe.

Palpitation.—Many cases begin with palpitation, and, while organic disease of the heart does not belong to aneurysm and the existence of it is the exception and not the rule, disturbed action is of frequent occurrence throughout the whole course of the affection. Palpitation is often paroxysmal or develops in certain postures. Thus the patient is often compelled to observe a particular posture, semi-recumbent and more or less right lateral, in prevention of palpitation and pain.

Pressure Signs.—As the aneurysm encroaches upon contiguous structures it shows pressure signs in symptoms indicating disease of any of the organs in the chest or abdomen. The most constant of these signs is that produced by pressure on the brachial plexus, causing brachial neuralgia, which shows itself in intense pain in the chest, radiating toward the

shoulder. These pains are much more frequent on the left side, and may be associated with formication, paræsthesia, and paretic conditions in the affected arm. Sometimes there is hyperæsthesia, sometimes anæsthesia dolorosa. Sometimes, especially when the ribs and vertebræ have been eroded, intense pain irradiates to the left arm. Hoarseness of the voice, or aphonia, may result from implication of the left recurrent laryngeal nerve. By reason of its anatomical situation the right nerve

FIG. 28.



Aneurysm of the ascending aorta (woman aged 30): *A*. Aorta: *B*, orifice of communication; *C*, aneurysmal sac: *D*, rib adherent to the sac; *E*, shrunken aortic valves (Ziegler).

escapes, though the right recurrent has been found affected in double aneurysms. Pressure upon a bronchus or upon the trachea produces dyspnœa, and many cases are announced or attended by attacks of dyspnœa of sudden occurrence. In fact, paroxysms of dyspnœa are wont to manifest themselves throughout the course of the disease. Extreme pressure may solidify the lung and give rise to bronchial respiration and bronchophony. Cough and hæmoptysis are not infrequent signs of aneurysm of the aorta. Pressure upon the superior vena

cava or upon the innominate vein may be made evident by a distention of the cervical veins and œdema of the face, neck, and upper extremities. Litten demonstrated a case in which an aneurysm of the ascending aorta had made compression upon the inferior vena cava. In this case there were six parallel rows of venous cords, as thick as fingers, which pursued a serpentine and tortuous course from Poupart's ligament to the level of the fifth ribs, and a bunch of large veins at the xiphoid cartilage. The pulsation of the tumor was so distinct and so exactly synchronous with the action of the heart as to illustrate the statement of Stokes, that it felt as if there were two hearts in the chest. The inosculation between the azygos and hemiazygos veins had con-

FIG. 29.

Aneurysm of the arch of the aorta. Woman aged 32: duration two years, dating from violent strain; tumor first visible eighteen months ago; has now reached the size of a small cocoanut; the surface is discolored; bruit inaudible over the surface; is distinctly appreciated when the bell of the binaural stethoscope is held in the mouth of the patient (Whittaker).

ducted blood from the inferior to the superior vena cava, and thus prevented ectasiæ and varicosities of the veins of the inferior extremities. Pressure upon the œsophagus produces dysphagia. Pressure upon the

pulmonary artery, which happens most frequently when the aneurysm is on the concave side of the aorta, produces severe dyspnœa, systolic murmur in the pulmonary artery, and attenuation of the second sound of the pulmonary artery.

Thus an aneurysm may compress the œsophagus, branches of the vagus, phrenic, intercostal nerves, branches of the brachial plexus, the spinal column, which it may erode to affect the cord; further, the intestines, liver, bile ducts, kidneys, ureters. The compression signs may be insignificant, or in other cases may be so pronounced as to overshadow all other signs.

Tumor.—External aneurysms are usually readily recognized by the occurrence of a tumor in the course of an artery. The tumor is soft, yielding, and may be entirely emptied by pressure, to renew itself by relief of the pressure. It is hard to improve upon the statement of Ætius, who said that the signs of an aneurysm are a "tumor, which may be large or small, without discoloration of the skin or pain, soft to the touch, having a loose, spongy feel, and yielding in such a way to the pressure of the fingers that it almost disappears, but returning again on the fingers being removed." Pulsation is distinctly felt in it and on all sides of it; that is, the tumor expands in every direction. The friction of blood, as it passes through the sac, develops a peculiar vibration or thrill, which is exceedingly characteristic. The thrill is described as a *frémissement cataire*, as it is closely simulated by the feel of the body of a purring cat. This thrill was noticed as long ago as the time of Petit, who used it as a point of differentiation between the true aneurysm of dilatation and the false aneurysm of extravasation or effusion. Petit noticed also the bruit characteristic of all aneurysms, with the statement that "when the ear is applied to the aneurysm by dilatation a noise similar to that occasioned by the passage of water through the pipes of a fountain may be heard."

The Condition of the Heart.—The heart itself is in no way of necessity involved, and any change in the heart structure belongs to complications. In no one of the 10 cases of sudden death from rupture reported by Draper was there any pronounced lesion of the valves of the heart. Twice there was a thickening of the valve, which still remained competent, and in 3 cases hypertrophy of the ventricle with or without dilatation. Stokes states that the heart is normal, as a rule, even in the presence of great aneurysms, and Suckling shows that hypertrophy of the left ventricle occurs only after affection of the aortic valves. But the whole heart may be dislocated by the pressure of an aneurysm at its base. As a rule, the heart is more loosely held in place, so that on account of elongation of the aorta the apex may be found or felt beating as far down as the sixth or seventh intercostal space, and as far back as the axillary line, especially when the patient lies on the left side. This point is especially emphasized by Curschmann as of value in the early recognition of the disease.

The pulse often furnishes positive indications in that it may be retarded, and an exaggerated difference in time may be appreciated between the apex stroke and pulsation at the wrist. If the aneurysm is seated in the ascending part of the aorta, all the pulses of the periphery, as compared with the apex stroke, are retarded. The most frequent

changes are retardation and reduction in volume up to disappearance of the pulse in the radial and carotid on one side. In aneurysm of the ascending aorta the carotid pulse may be later than the apex stroke. In aneurysm of the innominate the pulse may be slower in the right carotid and radial. In aneurysm of the arch of the aorta the pulse may be retarded in the left radial. In aneurysm of the descending aorta the pulse is retarded in the crural in comparison with the radial. Faint differences, it must be understood, may be appreciated only by the sphygmograph. The curve is less high on one side, and the second secondary wave is less pronounced. Thus in a case of aneurysm of the ascending aorta and the arch, described by Fischer-Dietschy and quoted by Litten, the radial and carotid pulses were considered alike until the sphygmograph demonstrated that the curve of the right radial was less high and the secondary wave was less pronounced. The right carotid, on the other hand, furnished a normal pulse. From these facts the conclusion was deduced that the subclavian had been implicated in the disease process, while the right carotid was intact. But these things were observed only with the aid of the sphygmograph. Finally, it must be remembered that the diminution of expansion of the peripheral arteries is not pathognomonic of aneurysm, since it may occur in other affections, and it should be known also that sometimes the very reverse relation to the rule prevails. Thus in a case reported by Blackman a large pulsating tumor lay behind the right sterno-clavicular junction. The right radial pulse was full and strong—the left, weak, faint, scarcely perceptible. The autopsy disclosed an aneurysm of the trunk of the innominate. The left subclavian was found in a state of degeneration and was compressed by the tumor.

FIG. 30.

Tracing from a case of aneurysm of the aorta (Musser).

The aneurysmal pulse curve itself shows three elevations, of which the first two coincide with the ventricular systole. Blood rushes from the ventricle by emissions into the sac, whose double expansion occurs so quickly as to be irrecognizable by touch. The third elevation coincides with the closure of the aortic valves, and is therefore a good sign of their normal action (Litten).

Fenwick and Overend constructed an apparatus for tracing the pulsations of an aneurysm on a revolving drum from a tube introduced into the œsophagus. But the method had to be abandoned on account of the straining and retching with alarming hemorrhage which the tube caused in one case. As so many accidents—sometimes fatal accidents—have occurred under its use, the soft œsophageal sound or tube should never be introduced in a case of even suspected aneurysm.

Besides differences in time, the pulse may present differences in force and rhythm, and irregularities and arrhythmias are common occurrences in the course of aneurysm of the aorta. These differences of all kinds, which may depend upon direct pressure or upon dislocation of the main

branches of the aorta, become most distinct in all cases when the arm is held up vertically from the body.

PHYSICAL SIGNS.—*Inspection* of the chest may in the first stages reveal nothing unusual, though, as a rule, the dislocation of the apex becomes visible as a pulsation lower than natural and nearer the axillary line. When the aneurysm reaches the wall of the chest, it makes itself manifest as a pulsation which is most commonly seen, because aneurysm of the ascending aorta is most frequent, in the second intercostal space to the right of the sternum. As the aneurysm continues to enlarge, the tissues of the wall of the chest, including the bones, suffer usury, and the aneurysm appears as a protruding, pulsating tumor, producing finally from its pressure a bluish or livid discoloration of the skin.

Neudörfer comments upon the fact as remarkable and inexplicable that the continued growth of the tumor leads to erosion of the sternum and the ribs and the displacement of the soft parts, so that the tumor appears as a strongly pulsating subcutaneous mass on the anterior surface of the thorax. It would seem to be easier to compress the lungs and soft parts than to consume the bony and cartilaginous wall of the chest, but experience shows the contrary. The lung tissue remains unchanged, while the bone yields before the aneurysm like wax. The chest can in no way restrain the growth of the aneurysm, which continues to grow outside the thorax, but only somewhat more slowly.

Even in the case of the popliteal artery, which is the most frequent of all, the bone suffers more than the soft parts. This destruction of bone by an aneurysm is not the consequence of an inflammation. There are formed no osteophytes in the vicinity to indicate irritation of the bone.

Palpation may appreciate more distinctly the change in the position of the apex and the pulsation of the aneurysm itself. Palpation reveals also, so soon as the tumor may be touched, a more or less distinct thrill, which is so characteristic as to be almost pathognomonic. Where the tumor can be encircled by the fingers or grasped by the hand it is seen that the pulsation is uniform—that is, that the tumor expands in every direction. The thrill is the vibration produced by the friction of the blood against the walls of the aneurysm, as it is often thickened and sometimes roughened by layers of coagulated blood.

When the aneurysm extends upward rather than outward, as is commonly the case in the affection of the arch of the aorta—this being the direction of the least resistance—the pulsation may be felt only in the jugulum. The patient is directed to extend the head backward, and the fingers are inserted behind the manubrium to feel it (Oliver). It must be admitted, however, that in certain cases pulsation may be appreciated in this way as the result of mere elongation of the aorta without dilatation.

Percussion shows dulness directly over the aneurysm, thus sometimes to the right of the sternum at the second interspace (aneurysm of the ascending aorta), sometimes at the level of the insertion of the clavicle (aneurysm of the innominate), sometimes under and above the manubrium (aneurysm of the arch). The absence of dulness does not exclude aneurysm, as the tumor may be so deep seated as not to reach

the surface at all. In fact, a circumscribed, tympanitic, or high percussion note may be the first objective sign of a deep-lying aneurysm of the aorta. Sometimes the dulness and pulsation disappear because the tumor changes its place. Small aneurysms, covered by the lungs, furnish neither dulness, pulsation, nor murmur.

Auscultation reveals a more or less distinct bruit from the rush of the blood through the dilated vessel. This sound is as characteristic as the thrill, which has been described as almost pathognomonic. The bruit is heard in the greatest intensity where the sac of the aneurysm comes in closest contact with the wall of the chest, usually at the seat of the greatest dulness and at the point where the thrill is most pronounced. Sometimes, as in the case of the aneurysm in the laundry-woman just cited, the bruit is not propagated to the outside, and can be heard only when the bell of the stethoscope is held in the mouth.

Sometimes the normal heart sounds can be heard distinctly in places where they are usually inaudible or feeble. Thus, it may be possible to hear the sounds, distinct and tolerably loud, in the left supraspinous region, because the dilated vessel, especially at the point where the arch of the aorta begins, has come to lie closer to the wall of the chest.

A peculiar extracardial murmur was recognized by Regaud in a case of a man aged seventy who suffered with dilatation at the origin of the aorta. In this case there could be heard, besides the murmurs of stenosis, a peculiar faint moist râle, which extended from the upper half of the sternum on the right side to the right axilla. The râles coincided with the systole of the heart and were independent of respiration. Regaud assumed in this case that there was an adhesion of the lung with the dilated aorta, and that alveolæ of the lungs were compressed with every action of the heart.

Aneurysm of the abdominal aorta remains longer latent, or is more apt to be misinterpreted in its symptomatology, than in any other part of the aorta. Pain is, as a rule, more diffuse and (with many exceptions) not so intense, and pressure signs have wider range in that the œsophagus, intestine, ductus choledochus, ureters, portal vein, neighboring arteries, inferior vena cava, spinal cord, and nerve trunks may be implicated to show signs in stenosis, obstruction, bile, and kidney colic, ascites, paræsthesiæ, paresis, and paralysis. The diaphragm may be pushed upward. Irritation of the vagus may produce paroxysms of vomiting (Traube). The pulse in the crural arteries may be retarded. The presence of a palpable tumor with distinct pulsation establishes the diagnosis. But pulsation in the abdomen is more deceptive than in the chest. Communicated pulsation from an underlying sound aorta to masses of fæces, accumulation of gas, or in thin people to the abdominal parietes, is very common, and pulsation, even with a distinct thrill, may be elicited at any time by mere pressure of the stethoscope.

Aneurysm of the pulmonary artery, which is extremely rare, occurs in consequence of arterio-sclerosis and of increased pressure in the course of the vessel—therefore usually as a result of extreme mitral stenosis. Skoda succeeded in distinguishing a case which had been mistaken for, or considered as, a mitral insufficiency. Extensive dulness, systolic murmur in the left ventricle, audible also over the right ventricle and at the base of the heart, with feeble heart stroke, dimin-

ished diuresis, hydrops, scarcely recognizable pulmonary tone, dyspnœa, and marked cyanosis, occurred in this case. The last three symptoms, which do not correspond to the picture of a typical mitral insufficiency, especially the extreme cyanosis with the scarcely audible pulmonary tone, should excite suspicions of an aneurysm in the pulmonary artery (Neusser).

Albu reports the case of a girl aged nineteen affected for years with tuberculosis marked by great shrinking of the left lung. There was displacement of the heart *in toto* to the left. There was active pulsation in the left second and first intercostal spaces, reaching to the left sternal border and to the left two fingers' breadth in front of the mammary line, propagated above to the right and below to the left. There was absolute dulness and a palpable fremitus corresponding to the pulsation, the dulness extending into that of the heart and lungs. There was certain hypertrophy of the right ventricle and probable hypertrophy of the left ventricle. The apex stroke was in the fifth intercostal space. A light systolic murmur was audible at the apex of the heart, and a systolic murmur over the region of pulsation, with very loud diastolic murmur; the second pulmonary sound was not increased. The aortic tones were pure. The pulse was small, but showed no other peculiarity. There was cyanosis of the lips and hands and some dyspnœa. The circumstances would not admit the diagnosis of aneurysm of the aorta, but could be interpreted as due to dilatation of the pulmonary artery produced by traction of the left lung.

Rupture is recognized by immediate collapse under the signs of profuse and rapid hemorrhage. Sometimes death is almost instantaneous. Especially is this the case where the aneurysm breaks into the pericardium or the vena cava. Pepper and Griffith found in the literature 29 cases of rupture of aneurysm of the aorta into the superior vena cava. When an aneurysm discharges its contents after erosion of the intervening structures into a large bronchus, death is usually rapid, under signs of suffocation with hæmoptysis. Sometimes the break is of such character as to permit only a slower oozing or escape of blood, when the patient may linger for a long time, becoming gradually more exsanguine. Sometimes, again, blood escapes only from time to time, as the orifice of the rupture may be closed by adhesions, etc. The sudden supervention of pleurisy or peritonitis would indicate escape of blood into the pleura and peritoneum; discharge of blood into the stomach is followed by hæmatemesis or enterorrhagia. Thus, Draper records a case of sudden death from thoracic aneurysm previously unrecognized, and mentions ten juridical post-mortems in which the cause of death was found to be due to rupture of aneurysm of the aorta. It is interesting to note that the rupture did not occur always in consequence of severe bodily strain or any particular motion, but that it happened in 3 cases in perfect rest, and that in 2 cases death did not follow immediately. The effusion took place usually in the pericardium, and the fatal termination appeared to be due not so much to the loss of blood as to interference with the action of the heart.

In other cases the effused blood was poured into the lungs, pleura, bronchus, or, as in the second case, into the abdominal cavity, penetrating the diaphragm. The aneurysms were by no means always very

PLATE V.

Aneurysm of the Basilar and Vertebral Arteries; small Rupture with Effusion into the Subarachnoid Space at the Base of the Brain. The Junction of the Vertebral Arteries is Absorbed in the Aneurysm. (Lehmann's Atlas.)

large; sometimes perforation occurred in relatively small aneurysms. Strange to say, external rupture is very rare.

Aneurysm of the arteries of the brain produces the symptoms of brain tumor. (See Plate V.)

Diagnosis.—The diagnosis of aneurysm of the aorta is determined by the attacks of pain, palpitation, and dyspnœa; by the dislocation of the apex of the heart, by the presence of a tumor which may be felt or seen, by the dulness to percussion, uniform expansion, pulsation, thrill, and bruit. Many of the symptoms of aneurysm are due to the mere taking up of space occupied by neighboring organs. Thus, there may be compression of nerves, of the vessels, of the trachea, of the bronchi, with the recognition of intra-thoracic, intra-abdominal, or intra-cranial tumors.

The recognition of causative conditions—syphilis, alcoholism, gout, an avocation of hard labor necessitating especially lifting or straining—is an auxiliary point of great value. It will be remembered, also, that the period about maturity is the age of aneurysms.

The situation of the tumor may indicate or exclude aneurysm of the aorta; thus a tumor in the median line or to the left of the median line does not point so strongly to aneurysm. In 32 cases of aneurysm of the aorta Sansom found pulsation in 25 to the right of the sternum, and in but 6 to the left of the sternum. In the majority of cases the maximum pulsation is found in the second intercostal space.

Pressure signs—as on the part of the œsophagus, dysphagia; on the vena cava, œdema; on the pulmonary artery, cyanosis; on the recurrent nerve, hoarseness of the voice or aphonia—may fix the character of the affection. Hoarseness, paralysis, and immobility of one (the left) vocal cord speak for compression of the recurrent nerve of the vagus; paroxysmal dyspnœa and dysphagia, for compression of other branches of the vagus or of the œsophagus. Paralysis of both vocal cords is more frequent in tumor of the chest than in aneurysm. Paralysis of the right vocal cord is against compression by aneurysm (Fränkel). Chaplin reported a case of aneurysm of the arch of the aorta in a man aged thirty-five, who showed the symptoms of paralysis of the recurrent of the left side, and who died of hemorrhage into the air passages. On post-mortem it was found that the aneurysmal sac had been entirely closed with layers of coagulated blood as completely as could have been wished.

One of the symptoms of thoracic aneurysm shows itself under minute perforation in the escape of blood as a repeated hæmoptysis, what Hampelen called the *habituelle prämonitorische*—hemorrhages along with local catarrh and irritation, which cause cough. Sometimes the diagnosis in these cases is only made by a post-mortem examination after rupture into the trachea, bronchi, pleural sac, or pericardium.

It may be understood that the symptoms may change under adhesion of the aneurysm or contraction of the orifice of the sac or thrombosis in the interior.

A case in which the organization and consequent reduction of a thrombus produced a change in symptoms was reported by v. Weismayer. The symptoms—pulsus differens, murmurs—changed from time to time, and it was seen upon autopsy that the thrombus at the

beginning of the subclavian artery, which had originally almost occluded the vessel, had become reduced under organization to such degree as to free the heart of the murmurs which it had caused.

Percussion reveals the tumor only after it has reached a certain size. But a mediastinal tumor will cause the same dulness.

A pulsation is necessary in the diagnosis of an aneurysm. In the absence of pulsation it is not justifiable to assume the existence of aneurysm. The expansile nature of the pulsation may be determined by covering the tumor with a piece of adhesive plaster slit in the middle. The pulsation of the tumor widens the slit.

Characteristic murmurs are not always heard in auscultation; moreover, murmurs may be caused by tumors. On the other hand, murmurs may be feeble and may be drowned under the natural signs of the heart.

The faintest murmurs may be recognized, Sansom says, by having the patient hold in his mouth the chest piece of the binaural stethoscope and close his lips over it. The observer may in this way detect a murmur communicated through the trachea too feeble to be transmitted through the walls of the chest. Systolic murmurs were heard in but 12 of 132 cases observed at the London Hospital, where the diagnosis was established in life.

The retardation of the pulse cannot always be regarded as a sign of aneurysm. The fact is, that when the pulse is re-established after ligation of an artery there is found no retardation in comparison with the other side, notwithstanding the fact that the blood must take the circuitous course of a collateral circulation to reach the ligated vessel.

Anderson declares that the physical signs of aneurysm of the aorta are most distinct when the aorta is affected in its ascending and the beginning of the horizontal portion of the arch, while the symptoms of pressure are more distinct when the aneurysm is seated in the descending portion, and especially when the sac springs from the posterior surface.

The diagnosis of aneurysm of the abdominal aorta is especially difficult when there is much panniculus adiposus or when there is meteorism of the intestines. Even when a tumor can be felt the nature of it cannot always be determined. And even after erosion the diagnosis is not absolute. A pulsating sarcoma may give rise to pulsations. On the other hand, an aneurysm may be lined with thick layers of fibrin which reduce the pulsations.

The Final Test.—Should there still be question of the nature of a tumor, all doubt can be dissipated at once by aspiration. Fürbringer decides his cases by puncture with the finest needle, finer than that which is used for acupuncture and galvano-puncture. As the needle penetrates, aspiration is practised, and, as arterial blood suddenly spurts into the instrument, the diagnosis is assured. With aseptic needles this exploitation is perfectly safe.

Differential Diagnosis.—Confusion with *abscess*, however frequent in the external arteries, is not common in the case of aneurysm of the aorta.

Stenosis at the aortic orifice may lead to a mistaken diagnosis, but the hypertrophy of the heart which occurs in this condition does not displace the apex to the same degree. Aortic stenosis shows also a

characteristic pulse, the so called wire pulse. Evidence of anæmia of the brain or of insufficient nutrition belongs to the defective blood supply of this condition. Both affections may show bruit and may show thrill, but aortic stenosis is not distinguished, as aneurysm sometimes is, by any accentuation of the second sound. The valve lesion is unattended with tumor—that is, with aneurysm—unless as a coincidence.

Hypertrophy of the heart is sometimes mistaken for aneurysm of the descending thoracic aorta, as the retrocardial pulsations of the aneurysm are communicated to the heart. But the neuralgic pains in the back, which occur eventually and are increased by movement of the body, retardation of the crural pulse, manifest collateral insufficiency in the chest wall in consequence of compression of the azygos and hemiazygos veins, dysphagia, especially in horizontal decubitus, soon decide in favor of aneurysm.

Bronchial asthma, paroxysms of which may be simulated in the varied symptomatology of aneurysm, may be separated by the history of the case, as asthma often dates from childhood, by the pronounced expiratory dyspnœa of asthma, with the presence in the sputum of spirals and eosinophile cells. The affection of aneurysm is more like an attack of cardiac asthma, with both inspiratory and expiratory dyspnœa, with the profuse expectoration of foamy sputum rich in cells, often in pigment cells (asthma humidum). The dyspnœa of aneurysm is most distinctly evoked by effort.

Mediastinal tumor or abscess may furnish dulness, but could not furnish the other signs, the pulsation and the thrill of aneurysm. In the rare case in which pulsation is felt because of superimposition it may be evident that the expansion is not uniform. Mediastinal tumor is more apt to show unilateral œdema (Ewald) and fulness of the small veins on the surface (Vierordt). Tumor has not the peculiar erosive properties of aneurysm.

Seat.—The diagnosis of the seat of an aneurysm is always difficult when the tumor occupies the base of the neck. It is sometimes impossible to decide whether the aneurysm lies upon the aorta or the innominate, the subclavian or the carotid arteries. In these cases the sphygmograph may be of great value, and the situation may often be determined by modifications in the peripheral circulation.

When an aneurysm is situated at the arch of the aorta the pulsation is double or may be double. On the other hand, when the aneurysm develops in the course of the thoracic or abdominal aorta the pulsation is always simple. The first pulsation, which is never absent, is the distention of the sac by the blood from the heart. The second pulsation, noticed at the arch of the aorta, results from the closure of the valves which return the blood. The second pulsation can be perceived only in that part of the aorta where the shock of the closure of the sigmoid valves can be felt, and in the part accessible to the reflux of blood from the carotid or subclavian arteries.

For the rest, the seat of the aneurysm is chiefly located by pressure signs.

PROGNOSIS.—The prognosis is always grave. The sword of Damocles is suspended by a hair over the head of the victim of aneurysm of the aorta. The sac may rupture at any time, especially under any

strain, at times even at perfect rest. Nevertheless, aneurysm even of the aorta is not necessarily fatal. Layers of coagulated blood, as stated, may line the interior or may even obliterate the sac with perfect *restitutio ad integrum*, "for the earthy matter of the blood," said Parè long ago, "being dry and thickened, adheres to the coats of the artery and to the parts that it occupies, thus to become bony and hard; and this by great foresight of nature sets a rampart of strong barrier, lest the hot and boiling blood . . . . should escape and pass out of the coats of the aneurysmal artery." Sutton reported the cure of an aneurysm of the arch of the aorta by rest and restricted diet, with the use of iodide of potassium and ergot. Quincke cites, in *v. Ziemssen's Handbook*, a number of cured cases, and Curschmann has lately added more. Lang, Lancereaux, and others report successful therapy with specific means.

Termination.—The fate of an aneurysm is fairly uniform. In most cases the sac continues to dilate, at times continuously, at times irregularly, often with long periods of quiescence or rest, corresponding in large degree with the force of the heart, but representing more especially advancing or arrested degenerative change. But sometimes the aneurysmal sac is obliterated entirely. This is more apt to be the case in the sacciform aneurysm, where the orifice of communication with the artery is small. The spindle-shaped is the most unfavorable, because of the difficulty of clot formation. Unfortunately, this happy result is not frequent, for, as was long ago remarked by Baillie, it is rare that the coagulum fills up the whole cavity in which it is formed. "Were this last circumstance often to take place, it would frequently become the natural cure of aneurysm and supersede the very painful means of a doubtful operation." Home and Hodgson believed it possible that the aneurysm by its pressure might obliterate the artery itself. This supposition is, however, impossible, because obliteration of the artery would release the pressure (Duplaix). Crisp believed that inflammation of the artery below the sac might close it. Cooper thought that the artery could be closed by compression of blood infiltrated into neighboring tissues. Hart maintained that fibrinous clots from the sac might obliterate the artery. It is certain that an aneurysm may terminate by suppuration or by gangrene in consequence of infection in the vicinity of the sac.

In certain cases the sac is actually so filled in as to leave a channel for the circulation of the blood. This interesting condition was noticed by Lancisi, who, in speaking of what he calls polypus incrustation lining the sac of an aneurysm, says: "For in an aneurysm that resembles a bag in shape the polypus crust lines the sides, so that the blood can flow through the centre of the artery, the circulation not being interrupted."

The vessel wall, as a rule, undergoes progressive atrophy and yields to the continuous pressure. Finally, there is left but one coat, and that becomes thinner and thinner until it bursts. The constant tendency is to expansion, attenuation, and final rupture. It is needless to state that any of the strains which contribute to start may finally break an aneurysm. Litten reports the rupture of a diffused aneurysm in the case of a man who tried to stop a horse. Coitus has been the cause of many fatalities in this way.

It is said that an aneurysm must continue to grow until it ruptures. This view is incorrect. Aneurysms may remain quiescent, may become smaller, may even entirely disappear. Such a spontaneous recovery is rare, but has been repeatedly observed.

Even aneurysms of the aorta need not necessarily prove fatal by hemorrhage. Aneurysm of the aorta may take life quite as frequently, if not more frequently, by pressure upon the vagus and phrenic nerves; by compression of the trachea and suffocation; by inflammation, thrombosis, and embolism; by interruption of the circulation; by coagulation in case a collateral circulation fails to develop; in the extremities by gangrene.

Absolute cure of an aneurysm is possible, but only at cost of the artery, which is rendered impermeable and is obliterated, so that the obliterated vessel ceases to be an artery. Curschmann observed several times resolution and entire dissipation of aneurysmal sacs which had advanced so far as to threaten to penetrate the skin. Whether or not this favorable result was the consequence of treatment with the sodium iodide and cold may not be so distinctly stated. In one case the ligation of the carotid was attended with the most favorable temporary results.

Duration of Life.—It is difficult to fix the average duration of life with aneurysm, as it is usually impossible to determine precisely the period of inception, but, dating from the diagnosis, Lebert establishes the average duration at fifteen to eighteen months. According to Puppe, the duration ranges from two months, the minimum, to twenty-five months, the maximum, with an average of ten and three fourths months. But in exceptional cases an aneurysm may be carried as long as ten to twenty years. Bostroem collected 117 cases, among which were 18 recoveries; also 150 deaths by hemorrhage. Blood escaped into the pericardial sac in 90 of these cases. Goupil saw such an aneurysm last eleven years, Peacock eighteen years. Bostroem believed that his case had lasted twenty-two years.

Prophylaxis.—Prophylaxis may be summed up in the avoidance of syphilis or in the long and thorough treatment of it, having been contracted.

TREATMENT.—The treatment of aneurysm is directed to the imitation of the process of nature in securing coagulation of the blood. The cardinal ends to be secured in aneurysm are retardation in circulation and lowering of blood pressure. These effects are principally attained by absolute rest, under which the activity of the heart is reduced, and by diminution of the nutrition.

Absolute rest in bed, best on a water cushion, for two to two and a half months has a remarkable influence in slowing the force and frequency of the action of the heart, and the occasional application of an ice bag or water-cooling apparatus helps to secure this object. Tufnell found in one case that the pulse in standing made 96 strokes, but sank after forty minutes of horizontal posture to 66—that is, 30 strokes in the minute—relieving the heart in twenty-four hours of 43,400 contractions.

Venesection fell into disuse on account of its danger, but has been recently renewed by Davison, who withdrew 700 to 900 grammes (22–

28 oz.), enjoined rest for several months, and limited the amount of drink. Venesection sometimes fails completely. Thus, Bret reported a case in which repeated venesection in no way prevented the rapid distention and final rupture of the sac.

To continuously retard and lessen the frequency and force of the action of the heart the patient is enjoined to observe the most absolute rest. Scarcely any excuse may justify an individual affected with aneurysm of the aorta in leaving his bed or in making any unnecessary effort. In this regard it must be seen that the bowels are evacuated regularly and without strain. They should be moved regularly, and are best evacuated by injections, or in robust patients by the use of salines or the infusion of senna, which lowers the blood pressure. The compound liquorice powder, one teaspoonful at bedtime, or a single tamarind, contains the virtues of a dose of senna. Gentle massage of the lower extremities and the abdomen dilates the vessels and lowers the blood pressure. Coitus must be refrained from.

Every imprudence in diet must be avoided. The patient should live as largely as possible upon fruits and fresh vegetables. Tufnell specifies a sparer diet as follows: breakfast, bread and butter two ounces, milk two ounces; dinner, meat two to three ounces, bread two to three ounces, milk or claret, two to four ounces; supper, bread two ounces, milk two ounces. Thirst is best allayed by ice pills. Should the patient become restless and irritable under this limitation, the quantity of food may be increased somewhat. In this connection, also, the patient should cultivate as much as possible under the circumstances tranquillity of mind, and there should be secured cheerful surroundings, which reconcile the patient to treatment by rest and partial starvation.

Murchison and Hilton Fagge, victims of aneurysm, continued to work to the last moment, but the chapter on aneurysm in the *Practice* of Fagge was written by Wilks.

There is no doubt, now, that limitation of the food and drink within certain limits lies in the direction of scientific treatment. Later, treatments after this method were adopted by Bellingham and Tufnell. Moxon, Vogel, MacKellar, and others have reported spontaneous cures in consequence of exhausting diseases attended with emaciation. It is, in a general way, emaciation and not anæmia which is to be considered the object in treatment of aneurysm by diet (Bäumler).

Symptomatic Treatment.—The effects of pressure in producing pain and interfering with the function of the organs are symptomatically combated. Thus pain, when not too severe, may be relieved by phenacetin, antifebrin, or antipyrin. The use of opium is to be delayed as long as possible. Sometimes the application of an ice bag or a belladonna plaster of sufficient size gives relief, or light compression may accomplish much. In one case recorded by Bäumler the application of a rubber belt lined with leather and cork and fastened with buckles secured dissipation of the pain and diminution of the tumor. The patient in this armor was able to ascend stairs and pursue his work. Such a support is especially indicated so soon as the aneurysm attenuates the chest wall. Attacks of angina are controlled by inhalations of amyl nitrite and the internal administration of nitro-glycerin. Shattuck especially recommends, in the relief of attacks of angina connected

with aneurysm, the chloride of barium, which is best given in pills of one tenth grain each three times a day after meals. The writer got some relief in this way in the case referred to elsewhere. Nervousness and sleeplessness may be subdued by the bromides—sleeplessness especially by trional, or, if necessary, by chloral. Palpitation may be relieved by the bromides or by the application of an ice bag over the region of the heart. Throbbing pain in the aneurysm itself is often quickest combated in the same way. Violent pains or palpitations call for and entirely justify the use of morphine, as tumultuous actions of the heart increase the difficulty and the danger.

Hemorrhage may be allayed by an ice bag applied to the chest, with the internal administration of morphine. External hemorrhage demands the use of compression and, if there is no fear of gangrene, of cold. Bäumler recommends as a styptic ferripyrin in powder or a 15 or 20 per cent. solution applied with cotton. Sometimes a venesection may relieve an internal hemorrhage or reduce a pressure in the sac.

Dyspnœa may demand the use of morphine. If there is compression of the trachea or paralysis of the recurrent, it may be necessary to do tracheotomy or introduce a long cannula.

Iodine.—In every case therapy is addressed to the diseased wall of the bloodvessel itself, and for this purpose resort is had to the iodides almost in routine treatment. In fact, as already remarked, the iodides are considered the remedies for the arteries. As a rule, the sodium is preferred to potassium iodide, because it is less irritant to the stomach and is said to better support the heart (Sée). There is, anyhow, a widespread belief among medical men that the potassium salts are more poisonous to the blood. The sodium iodide may be given in dose of 5 to 15 grains, three times a day, preferably in milk, before meals. Or, as stated elsewhere, all the good of iodine may be got out of the tincture, which does not irritate the stomach or degrade the blood. The tincture is best given in the dose of ten drops in a wineglass of sweetened water before meals. The free ingestion of any of the bland mineral waters flushes the remedy through the system and best prevents irritant or toxic effects. No mineral water is quite so good as milk when it can be obtained pure or if the water with which it is diluted be pure. The beneficent effect of the iodides in syphilitic arteritis can be readily understood; at least it is assumed to be understood from the known action of the iodides upon this disease. For the rest, the claim is made by Balfour that the iodides slow the action of the heart, lessen its force, and thicken the bloodvessel walls. Whatever may be the theory of its action, cases of cure under the use of it have multiplied to such an extent as to establish its value in therapy. Thus, to cite only one from a hundred authors, Keith reported a case of aneurysm of the arch of the aorta which was much improved under the use of potassium iodide in the quantity of 1200 grammes (37½ oz.) in ten months.

Local Treatment.—Broca long ago divided the numerous therapeutic procedures into two classes—agents which address the aneurysm directly, with the view of destroying it or causing it to disappear; and agents which address the aneurysm indirectly through the circulation, with the view of retarding it or of inducing coagulation of the blood in the sac.

Of chemical agents to secure coagulation, Wright recommended the calcium chloride, which was used by Shattuck and Wooldridge, and Haliburton tried nucleo-albumin—remedies whose effects could be in no way subject to control. The injection of nucleo-albumin has been followed by extensive coagulation in various parts of the body, with the dangers of fatal embolus. The only plan of treatment which could act in this way would be mechanical, either by retarding the current in the sac by giving the blood plaques numerous points of adhesion, or by mechanical injury of the intima to lead to the deposit of blood plaques.

These methods include venesection, cardiac sedatives (aconite, belladonna, veratrin), opening of the sac, extirpation, cauterization, application of stypties, of moxas, of refrigerants, caloripuncture, manipulation, acupuncture, direct and indirect pressure, flexion of joints, injection of coagulating matters, including foreign bodies, wire, watch-spring, silk thread, etc., galvano-puncture, and ligation of arteries before and behind the sac.

Filipuncture.—Stewart introduced fine silver wire, in one case 2½ feet, by means of two needles, and connected it with the positive pole under a current of 70 m. for one hour. This method was rewarded with recovery in the case of a patient aged twenty-five. Moore (1864) introduced foreign bodies in order to secure the deposit of fibrin, coiling in fine iron wire and introducing 26 yards. The coiling of the wire in the interior of the aneurysm favors the deposit of fibrin and prevents the dissipation of clots. White and Gould introduced into the sac of an aortic aneurysm 32 feet of steel wire. Bacelli (1873) used fine watch-springs, introducing seven such springs, each of the length of 50 cm. (in all 3.50 m.), through the cannula of a trocar. Death occurred in two days, while but little coagulation was found in the sac. Lépine used watch-spring, introducing it without a trocar. Ransohoff introduced 86 inches of silver wire; temporary improvement was followed by death in four weeks from hemorrhage into the right pleural sac. Lewis Bryant introduced organic matter, horse hair; Schrötter introduced silk threads; Murray, catgut. Temporary improvement was observed in some of the cases, with induration of the tumor. Verneuil in a discussion at the Paris Academy would limit this "treatment by filipuncture" to aneurysm of the abdominal aorta. Pringle reported a case of aneurysm of the abdominal aorta treated by the introduction of steel wire into the sac after laparotomy.

Needling.—In the modernized acupuncture Macewen transfixes the aneurysm with a needle and scratches its inner wall for ten minutes. The needle is then allowed to remain in the sac, but not longer than forty-eight hours. Under the irritation thus produced the whole sac thickens in the course of several weeks. As the operation is painless, it may be repeated from time to time.

Bignone treated a case after this method in a woman aged sixty-eight. Two needles were introduced November 30, December 4, 6, 14, January 10, and were permitted to remain twenty-four hours. By the middle of December the tumor had reduced one third. Pulsation had nearly disappeared. In the last operation there was the feeling of the puncture of a fibrous tumor with a cavity in its centre. Caselli

cured a case of aneurysm of the innominate in this way. This operation of needling has been done in our country by Bryant and others. It must be admitted, however, that Weir and Page, who treated a case of large aneurysm of the ascending aorta just above the heart, found upon autopsy that the treatment had not caused the formation of any white thrombus. Bäumler reports a case also of aneurysm of the innominate extending to the arch, with closure of the aorta, carotids, and contraction of the right subclavian, in which the right subclavian had been tied by Kraske, where this operation of needling according to Macewen's method was tried. Wyeth reported an aneurysm of the aorta treated by the combination of Valsalva's and Tufnell's methods and by teasing the sac wall with a silver pin. It is certainly easily done and without damage. It is better if the steel needle is not too thin (Bäumler).

Electro-puncture injures the wall of the sac, introduces a foreign body, and induces chemical changes in the blood, with the formation of oxygen and acid at the positive pole and of oxygen and an alkali at the negative pole. The decomposition of the salts which hold the fibrin in solution deposits fibrin about the nodule. All these factors contribute to the formation of a clot in the wall of the sac.

Numerous investigators have shown that the best method of using electrolysis consists in introducing only the anode into the aneurysm, as the development of oxygen is less in consequence of immediate oxidation of the metal, and the clot is therefore less spongy.

The cathode is applied in the form of a large moist plate on the surface of the thorax. The current is used in strength of 10 to 20 milliampères; Stewart used as high as 70. The sessions were from ten to forty minutes. The wound of the needle is dressed with iodoform gauze or with a piece of sticking plaster, cotton, and an ice bladder. The operation must be repeated in other parts of the aneurysm after longer or shorter duration, days or weeks. Dujardin-Beaumetz in France and Tillmans in Germany used galvano-puncture or galvano-lysis in more than 100 cases, but got good results—*i. e.* anything like reduction of the tumor, lessening of the pains—in only individual cases. Embolism occurred in but one case, that of Henrot. Tonoli reported the cure of an aneurysm of the ascending aorta marked by an extra-pericardiac ampulla on the right side by means of electro-puncture.

Ligation.—The really brilliant idea of ligating the vessels on the peripheric side—that is, beyond the aneurysmal sac—was conceived by Brasdor (1795), and was first executed by Deschamps (1815), and after him by Sir Astley Cooper. But Wardrop early in our century had generalized the method, and first expressed the view that the ligation of all the arterial trunks departing from the aneurysm was not necessary. This operation, the so called Brasdor-Wardrop method, has since been repeatedly done with perfect success in aneurysm of the trunk of the innominate.

The Brasdor-Wardrop operation was done successfully by Marriott in a case of aneurysm of the innominate of six months' duration in a man aged forty-nine. For a week after the ligation of the subclavian and common carotid the pulsation was as strong as before, and the tumor had not reduced. The patient was kept almost four months in

bed. It was only after seven months that the tumor began to grow harder and smaller. The patient was able later to do hard work, and at the end of two and a fourth years had perfectly recovered. Heath tied the common carotid in the case of a man aged forty-eight who suffered with neuralgic pains in the right side of the head, neck, and shoulder and chest for three or four years, at the end of which time there appeared a pulsating tumor in the neck. All the subjective symptoms disappeared and the tumor became smaller.

Nancrede advises, in thoracic aneurysm, distal ligation of the carotid and subclavian arteries first. Failing with these methods, resort should be had to needling. In abdominal aneurysm needling should be done first. Winslow collected 126 cases of peripheric ligature in the treatment of aneurysm of the innominate and aorta, and concluded from a study of these cases that simultaneous ligation of both the right common carotid and right subclavian furnished the best results. One third of the cases showed decided improvement, and in three of the cases an absolute cure was established by autopsy, the patients having died two or three years after the operation from other diseases. Finally, Fenger concludes from his studies that ligation of the right common carotid and subclavian arteries gives the best promise in aneurysm of the ascending aorta. In case of aneurysm of the horizontal portion of the arch one of the large vessels on each side may be ligated at one sitting, provided that both common carotids are not ligated on the same day. In aneurysm of the innominate artery the right common carotid and subclavian should be tied at once. If the tumor continue to develop to the right of the sternum, ligate the right vertebral artery. If the tumor develop to the left of the sternum, ligate some time later the subclavian artery. An interval of several months should always elapse between ligation of the two carotids.

Sometimes the effect upon the aneurysm is manifested only after the lapse of considerable time. Thus in a case of aneurysm of the innominate Marriott ligated the right subclavian and right common carotid, but noticed no effect for five months; at the end of which time the tumor began to diminish and to grow hard, so that the patient was able to return to the hardest kind of labor (shovelling coal from a wharf boat) in apparent perfect health (Fenger).

Astley Cooper (1817) first ligated the abdominal aorta for aneurysm in the groin. The operation has since been repeated a number of times without success. Finally, Loreta of Bologna succeeded in securing the occlusion of a large aneurysm in the case of a sailor in whom the dilatation had developed in consequence of a blow. The incision necessary to expose the aneurysm extended from the ensiform cartilage to the umbilicus, and the aneurysm when exposed was filled with forty inches of silver-plated wire. Pulsation ceased in twenty days, and the patient recovered completely in three months.

Ligation of the internal carotid has been recommended in the treatment of intracranial aneurysm. Pusey reported a case of this kind attended with success. Before such an operation should be attempted the ligation should be preceded by careful compression upon one or the other artery.

Compression with a tourniquet has been used with advantage in the

treatment of abdominal aneurysm, especially by Woirhage, who reported 9 cases, with 6 good and 3 bad results. The bowels are first thoroughly evacuated, and the tourniquet is applied to effect complete arrest of the circulation for four hours. If at the end of this time any good results are seen, the instrument is readjusted and the compression continued for another hour. Compression produces sharp local pain, sometimes also vertigo, syncope, and serious brain symptoms, probably through sympathetic reflex influences. Strong pressure upon the abdominal ganglia, the testicle, etc. may produce the same effect, but light remittent pressure is not painful. The patient presses the flat of his hand upon an aneurysm and feels relieved by it. So the use of a yielding elastic pressure is the best.

Continuous compression between the heart and the aneurysm may be tried in the case of the abdominal aorta. Murray of Newcastle succeeded in curing a case of aneurysm of the abdominal aorta in a man aged forty-six after compression of five hours under chloroform. Pulsation could not be felt in the lower part of the artery or in the femoral a year after the operation, though there was a satisfactory collateral circulation. Moxon and Durham of London succeeded in curing a great aneurysm of the aorta high up in the abdomen by ten and a half hours' compression by the Lister tourniquet. Compression of the abdominal aorta entails the danger of peritonitis and damage to compressed organs, the pancreas, duodenum, and solar plexus. Illustrative cases are reported by Bryant and Paget. In one of these cases the abdominal aorta was compressed twenty-four hours. The compression should therefore be done with great caution, and gradually up to the necessary degree to arrest pulsation; moreover, it should not be continued too long.

Weber gives the following statistics of cure of aneurysm by compression:

In 10 cases,	after	7 to 25	hours.
In 17 "	"	1 to 4	days.
In 15 "	"	5 to 9	"
In 11 "	"	10 to 15	"
In 17 "	"	20 to 30	"
In 18 "	"	30 to 60	"
In 11 "	"	more than 60	days.

It is seen thus that when regular, systematic pressure is kept up for some time it may be followed by a cure. Pressure is best made by permanent digital compression, which requires three pairs—that is, six intelligent men. It was Vanzetti who first of all observed that intermittent produced as good results as continuous compression.

The permanent application of cold, as well as the shorter application of high degrees of cold, is absolutely injurious, and may be dangerous by producing gangrene. Cold is exceedingly painful and distressing to patients, and may be used temporarily only as an analgesic.

V. Langenbeck claimed to have effected several cures of aneurysm by means of the injection of ergotin.

Aneurysms of arteries of the extremities, which are only mentioned here for the sake of completeness, may be cured by extirpation of the sac.

Extirpation of the sac of aneurysms was first practised by Gualtani one hundred years ago, but was displaced by the operation of Hunter.

After the operation of Hunter pulsation sometimes returns or the aneurysm bursts or suppurates. Kubler collected 40 cases of extirpation, of which 39 had a favorable termination. In 11 cases the aneurysm was non-traumatic. The fear of hemorrhage from ligation of an artery near the aneurysm on account of disease of the vessel wall is not justifiable since the days of antisepsis, as amputations in old people show that, even though the vessels are degenerated in a high degree, no hemorrhage follows from the ligated arteries. Moreover, ligation of an artery at a point distant from an aneurysm does not secure the sound arterial wall. Littlewood extirpated an aneurysm of the femoral artery and a considerable traumatic aneurysm of the popliteal artery, both with very good effect. They were both diffuse aneurysms and furnished a correspondingly bad prognosis. Erichsen considers amputation often the only rescue for such cases. Littlewood advocates extirpation in diffuse aneurysms, and considers it the best method in other aneurysms to prevent them from becoming diffuse.

To sum up the therapy, it may be said, considering the danger of all interference, that a case which is quiescent under a quiet heart and a proper hygiene may be let alone: *Quieta non movere!* But in a case which is progressing the surgeon should interfere and soon. For, as said Lancisi, the pioneer in this field of work, in his book *De Motu Cordis et Aneurysmatibus*, Rome, 1728, which, though posthumous, made an epoch in the history of medicine: "But the surgeon, being enlightened by anatomy, ought to be on the watch before the disease has openly and unequivocably shown itself; so that, if he were unable to prevent its occurrence, he might at least be enabled to guard against the very speedy death of the patient."

THROMBOSIS.

Definition.—Thrombosis (*θρόμβωσις*, becoming clotted) is the occlusion of a vessel by coagulation of its contents and thickening of its wall, which results, sooner or later, in complete interruption of the circulation. The process may occur in any vessel, including the heart itself, but is more common in the arteries and veins than in the capillaries.

Thrombi develop in consequence of diseased conditions of the blood and of the bloodvessel walls. Mere retardation of circulation results in coagulation of the blood, but it is questionable if the process of thrombosis ever occurs from simple mechanical stasis. In the production of thrombosis the retardation is itself the result of some diseased process.

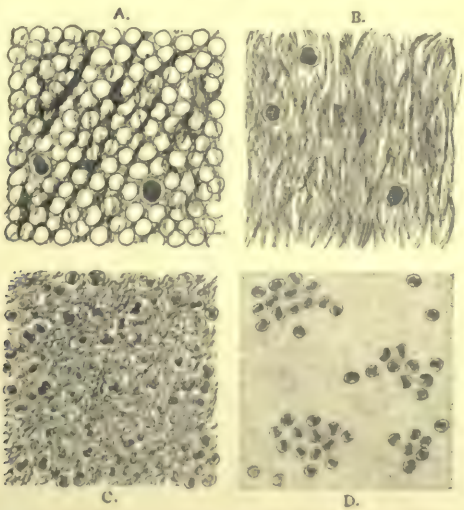
Two sources are given for the development of thrombosis: one the white, the other the red blood corpuscle.

1. Coagulation depends upon the contact of fibrinogenous substance with fibrin ferment in the presence of lime salts. According to Pekelharing, fibrin ferment is an organic lime combination, a nucleo-lime albumin. This combination gives up its lime to the fibrinogenous sub-

stance and changes it into fibrin. The fibrinogenous substance and the fibrin pre-exist in the blood in traces in the circulation, but they accumulate in quantity sufficient to produce coagulation only after dissolution of the leucocytes of the blood. Coagulation is therefore a process connected with disease and death of the leucocytes. The process of death begins in the blood so soon as it is withdrawn from the vessels, but anything which would favor the dissolution of the leucocytes, even in the course of the circulation, would cause coagulation of the blood. Thus the introduction of a large number of these cells from fresh pus or from cut-up lymph glands into the veins of an animal dissolves the leucocytes and, according to Groth, induces diffuse coagulation with immediate death. The introduction of a less number—that is, the dissolution of leucocytes in less degree—renders the blood in general incapable of coagulation. The dissolution of the hæmoglobin and the introduction of peptones produce the same effect, in that the blood afterward loses the property of coagulation. Hence the blood is fluid in the various fevers of sepsis, and, aside from local thrombosis, shows but little tendency to coagulate in the process of inanition.

Ponfick showed that the endothelium undergoes fatty degeneration in the grave infections, and is desquamated. The accumulation of these cells may start the process of coagulation. Every lesion of the wall of the vessel is marked by the accumulation of white blood corpuscles,

FIG. 31.



Agonal and post-mortem coagula: *A*, cruor clot; *B*, fibrin clot; *C*, leucocyte thrombus with fibrin; *D*, blood plaque thrombus with individual leucocytes (Schmaus).

which are subsequently converted into finely granulated fibrin. The dissolution of these corpuscles liberates the fibrin ferment which they contain, and the union of this ferment with the fibrinogenous ferment of the blood induces thrombus. Where the circulation is rapid the thrombus is composed more or less exclusively of the white corpuscles to constitute the *white* thrombus; when it is retarded the red corpuscles

are thrown from the centre to the circumference of the vessels, and enter into the formation of a thrombus to constitute the *red* or mixed thrombus.

Retardation of the circulation of the blood disturbs the disposition of its elements. Ordinarily the corpuscles occupy the axis zone, the serum occupies the border zone, while occasional white blood corpuscles roll slowly along the vessel wall. When the blood current is retarded the white corpuscles accumulate, to finally line the wall of the vessel. When the blood current is completely arrested the various corpuscular elements commingle.

Disease or injury of the wall of the vessel is repaired, or attempted to be repaired, by accumulation of blood plaques and colorless blood corpuscles, and thromboses are frequent in degenerations of the intima of the vessels. Anything which injures the intima—mechanical or chemical injury, inflammatory processes, atheroma, varices, and aneurysms,—anything which produces roughness of the interior may lead to thrombosis.

2. The first impulse in the process of coagulation comes from the red blood corpuscles, which in their disorganization furnish the granules called blood plaques. These blood granules are nucleo-albuminous constituents of the non-nucleated red blood corpuscles. They form in consequence of physical injuries which the blood suffers in contact with a foreign body, especially in the presence of adhesions. Thus the contact of red blood corpuscles with the surface of a vessel which has been mechanically injured or with a thread which has been drawn through a vessel brings about the disorganization of the corpuscles. The adhesion is to be looked upon as a trauma, which gives the first impulse to a change in the chemical relations between the cell and the plasma. The plasma, which has hitherto been a nutritive fluid, becomes a noxa to the red blood corpuscles. The nucleo-albuminous substance splits up and is dissolved. The degenerate cells lose their hæmoglobin and likewise suffer dissolution, and the firm union of the products of decomposition of the red blood corpuscles with the vessel wall is brought about by adhesive matter in the red blood corpuscles.

This process of dissolution and separation of the red blood corpuscles, erythrolysis and erythroschisis, not only brings about the formation of the blood granules and the thrombus, but causes also intra- as well as extra-vascular coagulation. The retardation of the current and mechanical circumstances, roughnesses, to which Eberth and Schimmelbusch have ascribed such an essential rôle in the development of thrombus, are, according to Wlassow, only auxiliary circumstances which determine the place of the deposit. But they are by no means necessary to the formation of the thrombus. The chief cause of the development of the white thrombus is the destruction of the blood corpuscles under the injurious action of the circulating plasma (Lubarsch). Damage to the wall of the vessel is followed by the exudation of fluid, which causes the destruction of a large number of the extraordinarily sensitive red blood corpuscles, and thus arises the nucleus or the centre of the white thrombus.

Forms or varieties of thrombus are described as *compression*, *stagnation*, and *marantic*.

A *compression* thrombus occurs in regions where the vessel suffers a sudden dilatation, as, for instance, in the heart in points above the venous valves, in pathological processes, varices, aneurysms. In all cases with the persistence of the condition upon which the process depends the tendency is to increase by the deposition of fibrin from the blood. Thus a thrombus which originates in a small vein in the extremities may extend into the inferior vena cava, and thence finally reach the heart itself (Ziegler).

The *stagnation* thrombus occurs in an interrupted or retarded circulation. But a mere retardation of the blood will not suffice alone to produce a thrombus. If a bloodvessel is tied in two places at a point where no branch is given off, with great care not to injure the walls, the blood will remain fluid in the region included between the ligatures for weeks. There must, therefore, be other factors, probably toxins, which kill the life of the blood. But stagnation or retardation itself in an indirect way interferes with the nutrition of the vessel wall.

ETIOLOGY.—Whether thrombosis of a vein is always of infectious origin may not be determined with certainty, but it is very probable that the so called *marantic* thrombus is not due to mere retardation of the blood current in consequence of weakness of the heart with a high degree of impoverishment of the blood, such as occurs in exhausting diseases, tuberculosis, cancer, etc., but that it is caused by matter of an infectious nature. Certain diseases produce toxins and reduce the antitoxic properties of the blood. The fact of the existence of these antitoxins is proven in the traumatic thrombosis in the veins of the puerperal uterus, which does not extend beyond the region of the placenta except in consequence of infection. In the case of a celebrated tragedy queen seen by the writer with Dr. Mitchell the entire left extremity became painful, œdematous, and blue. The swelling and discoloration extended to the veins of the neck, and a thrombotic deposit could be felt in the median vein of the neck, in the arcus venosus juguli. The pain and swelling were so great as to absolutely incapacitate any use of the arm, which necessitated the entire disbandment of the troupe. The patient had had a similar attack on a previous occasion in one of the lower extremities, and had become impressed with the statement of former physicians that she was affected with a peculiar vulnerability of the veins. No recent infection could be discovered in the history of the case, and the patient herself was inclined to attribute the accident to the strain of passionate declamation. In 19 cœliotomies von Strauch found three phlebo-thromboses of the left lower extremity, and attributed it to too extreme flexion of the thigh during operation. Sometimes in cases of apparent pure marantic thrombosis a small inflammatory dépôt, excoriation, or furuncle can be detected as the avenue of infection. Moreover, it is recognized that thrombosis of the veins occurs most frequently in consequence of certain infectious diseases—typhoid, typhus, influenza, diphtheria—and especially in pyogenic affections, as after operations with defective antisepsis.

In a case reported at Freiberg a robust girl had a moderate chlorosis, and fourteen days after a light follicular angina and thrombosis, first in one and then the other leg.

As diseases of the wall of the bloodvessel—arterio-sclerosis, amy-

loid degeneration, syphilitic infection, etc.—is produced through the medium of the vasa vasorum, it may be said that thrombosis in general results from some poisoning of the blood. Inflammation of the arteries themselves has been directly produced by the injection of pure cultures of micro-organisms into the blood by Gilbert of Lyons, and inflammation of the arteries as the result of infectious disease has been noticed in typhoid fever by Vulpian, Potain, Hayem; in diphtheria by Martin; in puerperal fever, measles, scarlet fever, smallpox, malaria, and rheumatism by Lancereaux and Mussy. The relation between arteritis and infection is no new discovery, for, as Lancisi remarked long ago, "Nor, indeed, can any one in his senses be surprised that if an eroding humor be mixed with the nutrient particles within the very texture of the artery, it may exert an injurious influence upon the structure of the vessel when we see that even the very dense structure of the teeth may be eroded by the action of deleterious liquids."

Thromboses of the heart are accumulations of coagulated blood in the auricles and the interspaces or recesses of the trabeculæ. As the coagulum increases it protrudes into the chambers of the heart to take up to greater or less extent the heart cavities. These accumulations, which were formerly known as polypi, are most frequent in the auricles. They may assume various shapes, may depend by more or less slender attachments and act as flaps or valves, or may be detached and remain separate as globular masses, especially in the cavity of the auricle. In the arteries the process begins in the quietest places, especially in dilatations, as is best seen in the layers which line the sac of an aneurysm. In the veins the deposits commence in the pockets of the valves. They may develop first in the capillaries, and extend thence to efferent veins, or, beginning in a small vessel, they may extend to larger veins. Marantic thrombi are found usually in the auricles, at the apex of the ventricles, in the pockets of the veins formed by the valves. (See Plate VI.)

Sinus Thrombus.—Thrombosis produces the most serious disturbances in the sinuses of the dura mater. These sinuses are particularly disposed to the process from the fact that they are tubes with fixed walls and angular lumen, and are traversed in places besides with a partition-like network. The thrombosis of inanition, the so called marantic thrombosis, is most common in the superior longitudinal and cavernous sinuses. Suppurative process in the bones of the skull, nose, orbit, and internal ear, or in the course of the veins communicating with these sinuses, as in erysipelatous or phlegmonous processes, may lead to the development of thrombus. Pitt recorded 22 cases of thrombosis in the lateral sinuses among 57 cases of fatal ear disease. The process, once commenced, shows the same disposition to extend here as elsewhere. Thus thrombosis of the cavernous and inferior petrosal sinus may extend to the internal jugulum and occipital veins. Suppurative thrombophlebitis may result in the development of abscess of the brain (Leube).

Cockel reported 5 cases of thrombosis of the veins in chlorosis, 2 of the brain sinuses, and 3 of the veins in the legs. In one of the 2 cases a thrombus deposited in distinct layers slowly blocked the transverse sinus, and the process of coagulation extended thence into the veins of Galen. The occlusion of these vessels developed hydrocephalic dilata-

PLATE VI.

Mural (Marantic) Thrombus of the Left Ventricle from a Case of Tuberculosis. (Lehmann's Atlas.)

tion of the brain ventricles. In the second case the process in the veins of Galen extended so rapidly as to result in hemorrhage and death before any distinct hydrops could develop.

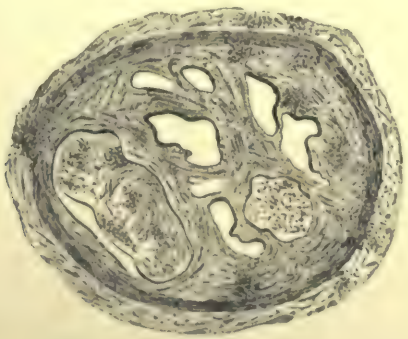
Renal Thrombus—Thrombosis of the renal veins has been observed, both as a compression and marantic process. Thus, Pollack saw thrombosis of the renal vein frequently in children affected with exhausting diarrhœas. Beckmann found it also in wasted children. Venous thrombosis of the kidney is tolerably frequent in amyloid degeneration. Occasionally phlebitis forms itself the cause of hindrance to the escape of venous blood.

According to Beneke, thromboses of the cavernous bodies of the penis may lead to gangrene or may be cured by a process of organization. Thrombosis from subcutaneous fracture has been most frequently observed in the thigh.

Peripheric Thrombus.—Hitherto the process has been described more especially as it affects the vessels of larger size, but peripheric thromboses are more common than is generally believed. Laache observed it in perityphlitis, pneumonia, especially often in influenza, acute joint rheumatism, chlorosis, and in advanced age without particular cause. Peripheric thromboses come mostly from some infectious process under the influence of a retarded circulation. In the acute infections peripheric thrombosis may occur also in the course of convalescence. In chronic diseases thromboses have been seen in the course of tuberculosis, syphilis, cancer, anæmia, rheumatism, obesity, severe strain. Ducket, Orlandos, and Annequin reported a number of cases of phlebitis with thrombosis of the veins after influenza.

The fate of a thrombus varies. Sometimes it becomes organized to reduce the tube to an impervious cord. The thrombus itself takes no part in this process, and the thickening is the result of proliferation of the endothelium. Thrombi which obliterate the calibre of the tube are known as obturating thrombi. Sometimes a thrombus shrinks and

FIG. 32.



Canalization of an old thrombus (Schmauss).

permits the blood to circulate again. The coagula may undergo calcification, with the formation of so called phleboliths and arterioliths, or suppuration to constitute purulent processes.

Symptoms.—The symptoms of thrombosis depend largely upon interruption of blood supply, and vary in their manifestations according to the region affected. They vary also according to the character of the thrombus and the rapidity of its formation. The signs of occlusion from the interruption in the circulation are œdematous swelling, phlegmasia alba dolens, and dilatation of collateral vessels with pain, tenderness, finally fever, which occurs also in cases of marantic thrombus. When the process occurs in the course of an infectious disease, or when the thrombus itself is composed of infectious matter, the condition may announce itself with rigors and fever. Thus thrombosis from suppurative processes in the middle ear develops with rigors, fever, pain in the head, delirium, etc.

Thrombosis of the cerebral arteries of slower development may produce various psychoses, alterations in disposition and character, paræsthesia and paralysis, hemiplegia. Pressure upon the cortex may develop somnolence, stupor, coma, or epileptiform and apoplectiform attacks, with psychical disturbance; or the symptoms on the part of motion may predominate to such a degree as to simulate progressive paralysis.

Thrombosis and embolism are the most common causes of softening of the brain. Thrombosis of a cerebral sinus may remain entirely latent or may show the signs later of meningitis in headache, vomiting, opisthotonos, convulsions, etc. Thrombosis of a cerebral sinus is best distinguished by the local effects of occlusion. Thus thrombus of the transverse sinus may be suspected in the presence of venous stasis and painful œdema behind the ears, at the mastoid process, and at the back of the neck. Thrombosis of the longitudinal sinus is marked by distention of the internal nasal veins, sometimes by profuse epistaxis, distention of the facial vein, of the veins of the temporal region, and in children by distention of the great fontanelles. Thrombus of the cavernous sinus is recognized by implication of the ophthalmic veins, œdema of the eyelids and conjunctiva, evidence of stasis in the retinal veins, œdema of the retina, exophthalmos, by paralysis of the muscles of the eye, and by neuralgia in the region of the first branch of the fifth pair. It is a matter of experience that thrombosis of the superior longitudinal sinus is, as a rule, of marantic, while that of the cavernous and transverse sinus is of inflammatory, nature, as excited by suppurative inflammation of the vicinity, most frequently in the case of the transverse sinus by tuberculous processes in the petrous portion of the temporal bone (Leube).

Thrombus of the femoral vein leads to white œdema, with distention and pain, which constitutes the well-known picture of *phlegmasia alba dolens*. This condition may occur in the course of any infectious process, but is especially liable to develop in the septic fevers of the puerperium. It shows itself in other infections more rarely—thus in about 1 of 100 cases of typhoid fever.

After review of 22 cases in obstetrical and gynæcological practice Mahler emphasizes among the symptoms of thrombus an entirely characteristic pulse and temperature curve, in that in the typical thrombosis the pulse runs up into a high curve, while the temperature remains perfectly normal. The increase in the pulse curve persists while

the temperature continues in its old course. In the presence of œdema, or when a thrombotic cord can be felt, or in the presence of symptoms on the part of the lungs, the pulse reaches its highest point, and occasionally the temperature at the same time reaches its maximum. But while in the course of the next few days the temperature falls, the pulse remains high for days. Wyder too remarks upon the fact that thrombosis of the internal veins is often signalized by rapid increase in the pulse in the first days after the operation, during a condition of relative health and under normal or nearly normal temperature. Mahler mentions as a further symptom headache, which often occurs early, together with the well-known lancinating, boring pains in the course of the veins in the leg. The writer has verified this condition of the pulse and temperature in a case of syphilitic thrombosis of the leg of quite sudden development, in connection with an obstinate ulcer on the shin.

Diagnosis.—The diagnosis of thrombosis rests chiefly upon the fact that the condition is found in connection with some infectious disease or with some of the circumstances which favor the development of arterio-sclerosis. Thrombus of more or less acute development occurs in puerperal fever and dysentery, especially in the lower extremities, and suppurative processes, especially in the head. Marantic thrombus shows itself after long-standing wasting disease, Bright's disease, carcinoma, tuberculosis, etc., or in inanition from any cause. Sometimes a thrombus may be felt, as in the femoral vein, as a hard cord painful or tender to pressure, or the condition is revealed by the evidence of the interruption of the circulation by stasis, œdema, coldness of the surface or cyanosis, sometimes by gangrene.

Thrombosis of the pulmonary artery announces itself with sudden intense dyspnœa, cyanosis, spitting of blood, sometimes with sudden death. Thrombosis of slower development shows itself oftenest in the brain, with signs of gradually interrupted circulation, eventuating in softening of the brain. The condition may be recognized here, too, by the age of the individual, by the precedence of causative factors, age, syphilis, alcohol, by long-continued depressing mental emotions, sometimes by the evidence of arterio-sclerosis elsewhere, by changes in disposition, paresis, etc.

Prognosis.—The prognosis depends upon so many factors, as the age of the individual, the character of the cause, the completeness or incompleteness of the occlusion, the region affected, that it may be made only in an individual case. Thrombus of the femoral vein, a most common site, has, as a rule, a favorable prognosis. Thrombus of the pulmonary artery is always grave, and often immediately fatal. Thrombosis of the cerebral arteries is usually slowly progressive and ultimately fatal. A suppurating thrombus disseminates the sepsis from which it develops.

Thrombosis is not only dangerous in itself, but also in its consequences, in that from the thrombotic mass particles may be detached and carried away by the circulation to be deposited in distant organs as emboli, with their peculiar consequences.

The syphilitic process is slow, and retrograde changes usually set in, marked by mucous softening and dry necrosis, to constitute the syphi-

litic gumma. Entire resolution may occur in the earlier stages, or a fibrous metamorphosis may permanently contract or obliterate the lumen of the vessel.

Prophylaxis.—The best point in prophylaxis is found in rest, which in the puerperal patient should be absolute—rest in bed in the horizontal posture. The pulse offers the best criterion of the time when a patient who has shown symptoms of thrombus or capillary lung embolism may leave the bed, as a fall in the frequency of the pulse indicates improvement in the circulation under absorption or organization of thrombi or emboli.

Treatment.—The treatment of thrombosis is a matter of address to the originating malady and to the relief of consequences. The process may be prevented or postponed for a long time by removal of diseased centres, by the neutralization of toxins, and by improvement of nutrition, especially by rest in bed.

When a vessel has become blocked effort is made to establish collateral circulation. In the case of the femoral vein the leg is supported upon an inclined plane, that the return flow of fluid may be favored. The limb should be elevated, sometimes as high as at right angles to the body. The arm, for instance, may be suspended from the ceiling or from a rope across the bed-posts. The leg is better supported upon an inclined plane, which may be readily improvised in any house with the leaf of an extension table resting upon a chair at the foot of the bed. Where the patient suffers intolerable pain the limb may be let down for a time, and the position resumed with the relief of the pain. Under this treatment the œdema and livid discoloration are wont to subside in the course of a few days. A collateral circulation is most rapidly established in this way. No other treatment compares with the elevation treatment. The patient should remain perfectly quiet from two to three or four to five weeks.

Attempt is made to secure absorption by the application of iodine in the course of the femoral vein. The pain of distention may be relieved by the application of a very light bandage. Anything like real compression is dangerous and may eventuate in gangrene. Thrombosis of the renal vein may be relieved to some extent by stimulating the blood to the surface, as by hot baths. Thrombosis of the pulmonary artery demands absolute rest, best in the semi-recumbent posture, with abundant ventilation of the lungs with fresh air, and the use of heart stimulants, caffeine, nitro-glycerin, digitalis, etc. In thrombosis of the cerebral arteries the patient should have the benefit of the iodides.

In avail of the well-known property of leeches in preventing coagulation the suggestion has been made to use infusion of leeches in treatment. The leech infusion does not in any way change the white and red corpuscles, and its effect does not depend upon a conservation of the blood plaques, which make their viscous metamorphosis just as before, but upon a destruction of the fibrin ferment. The experiments of Eguet under Sahli in this regard resulted in the following conclusions: (1) Infusion of leeches prevents the formation of thrombi about foreign bodies in the blood of rabbits. (2) The least dose which must be injected is the infusion of one leech to 55 c.c. blood. (3) The duration of the effect is less under good action of the kidneys. Making

an estimate for man from these results, it is found that a man weighing 130 pounds would require an infusion of eighty to ninety leeches in order to prevent the blood temporarily from forming clots. But as human blood is much poorer in fibrin than rabbit blood, it is probable that a less number of leeches would suffice. The suggestion is as yet purely theoretical, and is probably impracticable.

The treatment, for the rest, is purely symptomatic. The general strength may be supported with iron, quinine, and arsenic; local pains may be relieved by the salicylates, phenacetin, lactophenin, or, if necessary, by morphine. Warm baths may suffice to allay sleeplessness, nervousness, and lighter pains.

The sudden occurrence of colicky pains or a bloody diarrhœa, collapse, or peritonitis indicating thrombus or embolus in the domain of the mesenteric vessels may call for surgical intervention. A case of this kind in which there had occurred infarction from thrombus of a mesenteric vein in a man aged twenty-five was rescued by Elliot by resection of four feet of the intestine. Recovery was perfect. Watson advocated laparotomy and resection of all necrotic portions of the intestine under the use of the immediate suture and the opening of an anus preternaturalis, which may be closed later.

EMBOLISM.

Definition.—An embolus (ἔμβολος, a peg, a stopper) is a block of matter projected into the circulation, as the name implies, detached from some central trunk, usually a thrombus, to be arrested finally at a narrower part of the vascular tree. The effect of this occlusion, interruption of the circulation, and subsequent change in the region supplied by the vessel constitutes the process of embolism.

Emboli are distinguished as *direct*, *recurrent*, and *crossed* or *paradoxic*.

Emboli which follow the regular course of the circulation, as into the arteries of the body from the left heart (including the pulmonary veins), from greater arteries into smaller, from the right heart (including the veins of the body) into the pulmonary arteries, from the radicles of the portal vein into the ramifications of the liver, are known as direct emboli. Emboli which from their weight pass against the current, as is the case sometimes with parasites and parenchymatous cells, in the sluggish circulation of the veins, are known as recurrent or retrograde emboli; while emboli which pass the nearest vessels by means of some direct intercommunication without intervening capillaries, as in the derivative circulation of the lungs and liver, or which through an open foramen ovale traverse the heart itself, are known as paradoxic emboli.

The crossed thrombus or embolus is a rare process in comparison with the frequency of the persistence of the foramen ovale, though it may certainly play a rôle in the dissemination of both bland and infected emboli of parenchymatous cells and tumor elements, and probably plays a considerable rôle in the dissemination of tuberculosis in acute miliary tuberculosis, in tuberculosis of the thoracic duct.

Constitution of Emboli.—Masses of fibrin (vegetations) detached from the mitral valve or atheromatous particles or plaques (chalk) from the aortic valve constitute the most frequent deposits in the course of the great circulation. Particles of neoplasms, cancer, sarcoma, the deposits of syphilis, parasites, echinococcus, cysticercus, make the materials of emboli much more rarely. Litten once saw perfect closure of two chief branches of the pulmonary artery by living echinococci; further, a dead echinococcus cyst in the beginning of the upper mesenteric artery, likewise blocking it completely; behind it a hemorrhagic infarction of the wall of the duodenum. Rare matters are globules of fat from the bone marrow after fracture, or from the subcutaneous and submucous tissue, from injury or from convulsions or contusions, as in the pelvis in labor.

Fat embolus may be derived, besides, according to Ribbert, from inflammation and injury of the bone marrow, from inflammation of the subcutaneous and pelvic fatty tissue, from injury and necrosis of the liver, from thrombi which have undergone fatty degeneration, and from mere violent agitation of bone without any fracture. Thus in rabbits under narcosis a series of short strokes with a wooden instrument upon both tibiæ suffices in the course of one to two minutes to produce a moderately high degree of fat embolism.

Emboli of parenchymatous structure deserve special mention. Thus, Aschoff found emboli in the capillaries of the lungs which consisted of giant cells with multiple nuclei, and which seemed to be derived from the bone marrow and from the spleen and lymph glands. They were seen especially in the case of burns attended with destruction of the red blood corpuscles and after poisoning with pyrodin.

Lubarsch distinguishes three kinds of embolism of parenchymatous cells—the liver cell, the placental cell, and the bone marrow giant cell. The liver cell embolus results from detachment in consequence of trauma of the liver and separation in the course of infectious diseases. These cells have been found in the lungs, and more rarely in the brain and kidney veins, transported from the liver in cases of eclampsia during puerperium and in chorea. Liver cell embolus has been observed also in diphtheria and in abscess of the liver. Transported liver cells may live as long as three to ten weeks, but they never undergo proliferation.

Placental cells—that is, giant cells from the decidua—have been found transported in cases of eclampsia and chorea, and cells from the bone marrow in cases of fracture of bone in tubercular hip-joint disease and after resections. All these emboli perish in the course of a few months without the slightest trace of organization or proliferation. The placental cells perish quicker than the liver cells. These cell emboli are not the causes, but are the effects, of the convulsions in eclampsia and chorea gravidarum. Bone marrow cells have but little property of inducing coagulation.

Philippson observed in the course of a case of leprosy an acute eruption of erythematous spots which was found under the microscope to be caused by embolism with the lepra bacilli. Some of the capillaries were filled with bacilli. So the typhoid bacillus has been seen in the roseola of typhoid fever.

Fat embolus is derived almost exclusively, as the result of injury,

from the bone marrow, much more infrequently from subcutaneous tissue, liver, brain, or thrombi which have undergone fatty degeneration. Ribbert has shown, as stated, that fat may be absorbed as the result of concussion without fracture. Fat emboli come to be deposited for the most part in the capillaries of the lungs, but minute masses may be driven through the lungs and disseminated over the body.

Still more infrequent are the so-called air emboli, which result from the aspiration of air into open veins after operations especially about the neck, in the uterus after labor, and from air which has been rendered free in the blood itself in sudden diminution of pressure, as on the sudden return from compressed air in caissons. As curiosities in this connection may be mentioned the air vesicles which develop from the action of chemical poisons, diazobenzol, or from gas-forming micro-organisms.

Air emboli may be literally sucked into the circulation in the course of operations about the neck or after exposure of the great veins in the uterus. Heuck demonstrated such a case in labor with collapse and sudden death. The post-mortem made two hours after death showed air in the right jugular vein, in the right spermatic vein, and in the right heart. It was a case of central placenta prævia, with version by the feet, and it was thought probable that the insertion of the child's leg between the wall of the uterus and the detached placenta forced air into the lumen of the veins. Three cases of air embolus in placenta prævia were reported from the clinic of Olshausen in the last six years.

Site of Deposit.—Emboli may be deposited anywhere in the course of the circulation, but they are found most commonly—in fact, more often than in all other places together—in the course of the pulmonary artery, because of the frequency of thrombi in the peripheral veins and of thrombosis in the pulmonary artery itself as the result of arterio-sclerosis, in consequence especially of the strain of lesions of the mitral valve. Emboli are found next in frequency in the renal and splenic arteries, the arteries of the brain (fissure of Sylvius, corpora callosa, basilar arteries), the superior and inferior mesenteric arteries, the cœliac arteries. Emboli are rare in the arteries of the extremities, but if found at all are more common in the brachial artery.

Wherever lodged, an embolus always occludes the vessel. The occlusion may be incomplete at first on account of the size of the embolus or the shape (flat or angular chalk plate, etc.), but deposits of fibrin soon increase the size or fill out the interspaces to entirely block the vessel.

Emboli usually lodge in the course of a vessel at points of bifurcation, and often in such a way as to ride upon the spur or projection which marks the division of the artery. The embolus may then project into both branches—as Litten says, "something like the roots of a double molar tooth."

The effect of an embolus depends upon the constitution of the embolic matter itself and the character of the vessel which is blocked. Occlusion by masses of fibrin, chalk plates, etc. is limited to purely mechanical effects. Emboli which are composed of infectious matter distribute or disseminate the particular infectious process, often with the development of metastatic abscesses.

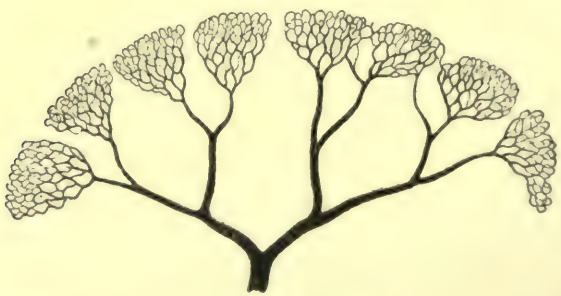
The first effect of embolism in all cases is arrest of the circulation, and the consequences of this effect in the domain of the vessel will depend altogether upon the possibility or impossibility of blood supply from other sources. If there is or can be no such supply, the tissue remains anæmic and pale and the affected region constitutes what is known as white infarction. Certain arteries divide dichotomously and

FIG. 33.

Schematic representation of an artery, showing abundant anastomoses (Schmauss).

form the exclusive supply of definite regions. Other arteries inosculate freely with each other, so that the tissue lies imbedded in a rich network of vessels. Cohnheim called the first set end or terminal arteries. Such end arteries are found in the kidneys, spleen, lung, parts of the brain (central ganglia, corpora striata, thalami optici), heart muscle,

FIG. 34.



Schematic representation of terminal arteries, showing but few anastomoses (Schmauss).

tympanum, and retina. The radicals of the portal vein in the liver are also anatomically terminal arteries, but, as every lobule is likewise supplied by a branch from the hepatic artery, the purely mechanical effects of closure of the radicals of the portal vein are scarcely ever seen in the liver (Sehrwald).

The change which takes place in the tissue under embolism depends upon the distribution of the vessels. In many organs the condition is always hemorrhagic, in others always anæmic, in still others it is variable. Where the collateral circulation is small the tissue remains anæmic; where it is abundant the tissue is hemorrhagic.

Infarction.—Dépôts of softening in the brain are always anæmic, and, though small capillary apoplexies may occur at the cortex, the typical hemorrhagic infarction is very rare. Infarctions of the kidneys are also anæmic, likewise of the retina after occlusion of the arteria centralis retinæ. Infarctions in the stomach and intestine are usually hemorrhagic because of the abundant anastomosis of the smaller branches. For the same reason infarction occurs in the stomach and intestine only under occlusion of the chief trunks. In the spleen infarctions are now hemorrhagic and now anæmic.

The tissues in the domain of the supply become at first anæmic, but the whole region is soon filled with blood by regurgitation from neighboring capillaries. Blood escapes also by diapedesis through the capillary walls to reach the surface with the secretion or excretion of the organ affected.

Finally, the whole tissue becomes infiltrated with blood, and appears as a dark red or black cone-shaped mass whose base is at the periphery of the organ and whose apex is at the site of the embolus. This condition represents the hemorrhagic infarction.

The older observers believed that infarction never occurred in normal lungs, but was always the result of interstitial pneumonia, heart failure, and bronchitis, which led to peribronchitis vasculosa. They believed that the hemorrhages arose from newly formed vessels in this process; the occlusions were therefore not emboli, but thrombi. Gzell subscribes, however, to the view of v. Recklinghausen and Willgerodt, that in man pulmonary infarctions are of embolic origin. Thrombi developed in the course of one to three days were introduced into the jugular vein of a second rabbit. The results of these experiments were not different from those produced by paraffin plugs, 1½ to 2½ c. long and 2 to 4 mm. thick. The picture of necrosis much predominated over that of hemorrhage. Thus in the development of pulmonary infarction in man there must be, besides the occlusion of the artery, an adjuvans to account for the origin of hemorrhage.

Collateral Circulation.—The fate of the part of the organ thus affected will now depend—aside from the character of the embolus—upon the possibility of the establishment of collateral circulation from the surrounding capillaries. This circulation is secured not so much by reflux from the veins, as Cohnheim believed, for Litten showed that hyperæmia and hemorrhage occurred in even greater degree when both artery and vein are ligated, but that there is no hemorrhage when all the vessels are separated except the renal vein, so that the only possible supply is the collateral circulation. Zielenko had already reached this conclusion in his studies of the circulation in the web of the frog's foot, and Scossuchin found hemorrhage by diapedesis only in the periphery of the embolized area and in the surrounding zone of collateral hyperæmia. Welch and Mall confirmed these conclusions, with the demonstration further that when the capillary circulation sinks below a certain point hemorrhagic infarction does not occur.

But arteries which are not terminal arteries in an anatomical sense may nevertheless show the same effects by reason of the few anastomoses which they receive, or by reason of the fact that the communicating vessels may themselves become blocked either by emboli or as

the result of arterio-sclerosis. Regions supplied by arteries with abundant anastomoses, where collateral circulation can be easily established, as in the skin, the muscles, various glands, or in the circle of Willis at the base of the brain, show little or no disturbance.

Embolism of the brain is usually derived from disease of the heart. Thus, Savaliew found heart disease in 89 per cent. of 191 cases. But other conditions—chlorosis, anæmia, leucæmia, scorbutus, syphilis, cancer, or tuberculosis—may lead to embolism of the brain. Brain embolism occurs preferably in the earlier decades of life, and more frequently in the female sex. An embolus detached from the heart finds its way most readily to the left carotid artery, because this vessel is given off at an acute angle from the aorta—more, therefore, in the direct line of the circulation. For the same reason the embolus passes most directly to the artery of the fissure of Sylvius and lodges in its cortical branch. This branch supplies part of the anterior and the whole of the posterior central convolution, the third frontal convolution, the superior and inferior parietal lobes, the island of Reil, the neighborhood of the fissure of Sylvius, and the third temporal convolution. The internal branch supplies the lenticular nucleus, part of the optic thalamus, the external and internal capsule, etc.

Metastases.—Infected emboli develop, in addition to the mechanical disturbances, chemical and pathological processes of much graver import. Thus fragments of particles of malignant neoplasm, carcinoma, sarcoma, set up new growth at the site of deposit, and micro-organisms develop colonies which further disseminate the original disease. The processes of sepsis are especially developed in this way, and the secondary symptoms often overshadow the original disease.

The course taken by metastases is through the lymph or bloodvessels, and observation shows that carcinoma prefers the lymph vessels, while other tumors, especially sarcomata, prefer the bloodvessels. Thus metastases occur (1) through capillary emboli, (2) through paradoxic emboli, (3) through venous retrograde emboli, (4) through retrograde lymph transport. The chief factor in the development of metastases is the constitution of the structure. Müller found in 521 cases of carcinoma metastases in 47.2 per cent., and in 102 cases of sarcoma metastases in 63.7 per cent. It is the especial quality of the tumor—what Hansmann distinguished as the anaplasia of the cells—and the rapidity of proliferation which leads to the formation of metastases. Nevertheless, benign growths also undergo metastases. Thus to the older cases of metastasis of simple gelatinous matter from goitre and from enchondroma reported by Cohnheim, Neumann, etc. are added in recent times the metastases of uterine fibroids—one by Klebs somewhat questionable, one unquestionable by Krische and Orth, with extensive metastases in the brain. Zahn has lately described cases of metastases of tumors in the stomach, ovary, and tonsils, and curiosities were reported by Schäper of metastases of a primary cancer of the lung into a uterine fibroid, and by Lubarsch of a papillary cysto-sarcoma of the right ovary with numerous small metastases into a large fibro-myoma of the left ovary (Lubarsch).

Symptoms.—The symptoms of embolism depend upon the character of the originating malady, upon the constitution of the embolus itself, and upon the interruption of function in the affected organ. Thus

thrombotic processes in the veins of the body give rise to the signs of pulmonary embolism. Marantic thrombi from the femoral vein, the saphena, internal spermatic, the plexus of veins in the pelvis, furnish the most common source of these emboli, while thrombotic processes in the pulmonary veins and left heart supply the emboli for the arteries of the great circulation. The process develops in general especially in connection with sudden movement or agitation of the body, as in excitements of the heart which follow the first efforts in convalescence, in arising from bed, the act of dressing, straining at stool, etc.

Embolization of terminal arteries is attended with the most disastrous results, and the sudden arrest of the blood supply may be marked by severe pain, tremor, spasms, or convulsions, or from reflex action under the extreme irritation by chill, fever, vomiting, and loss of consciousness.

An embolus of whatever character may show, as stated, its immediate effect in chill, fever, pain, hemorrhage, and arrest of function. The merely mechanical—or, as they are often called, bland—emboli may show no other evidence, and these symptoms may disappear in a short time with the establishment of collateral circulation, as in the case of the extremities, or they may be attended with the most disastrous results possible—cyanosis, dyspnœa, and suffocation when in the pulmonary artery, or hemiplegia and softening when in the vessels of the brain. The existence of the bland embolus may be surmised or established by the character of the originating thrombus, as in the case of simple verrucose endocarditis or local deposit in arterio-sclerosis. Infective phlogogenic emboli arise from infected dépôts or centres, as, for example, from the veins of the pelvis in puerperal infections, from the prostate glands in gonorrhœa, from the veins of the intestine in dysentery, etc. The character of the embolus may be determined by the fact of the existence of some septic disease. Infective emboli are distinguished also by the fact that particles detached from them penetrate the capillaries to produce metastatic abscesses, or pass through them to be distributed anywhere over the body.

The *fat embolus* is noticed after fracture or after laceration or comminution of the fatty tissue. The globules are seldom of large size, so that fat emboli usually come to lodge in the capillaries of the lungs, where they show the symptoms of interruption of the pulmonary circulation in dyspnœa, œdema, and sometimes with coma. As the fatty matter is not detached suddenly *en masse*, but gradually in the form of minute globules, the process of embolization is slow, and the symptoms on the part of the lungs show themselves in the course of several hours or days after the liberation.

An illustrative case of this kind the writer saw with Dr. Helm of Peru, Ind., who had established the diagnosis, in the case of a man who had suffered fracture of the leg, and in the course of six weeks was seized with the symptoms of pulmonary embolus, marked by a sudden attack of cyanosis with great anxiety, followed by bloody expectoration. This attack subsided in the course of a few hours and the patient seemed in a fair way of recovery. But these symptoms repeated themselves in the course of a week, and again subsided in forty-eight hours, to leave, however, a slight rise of temperature which marked an infarction of the

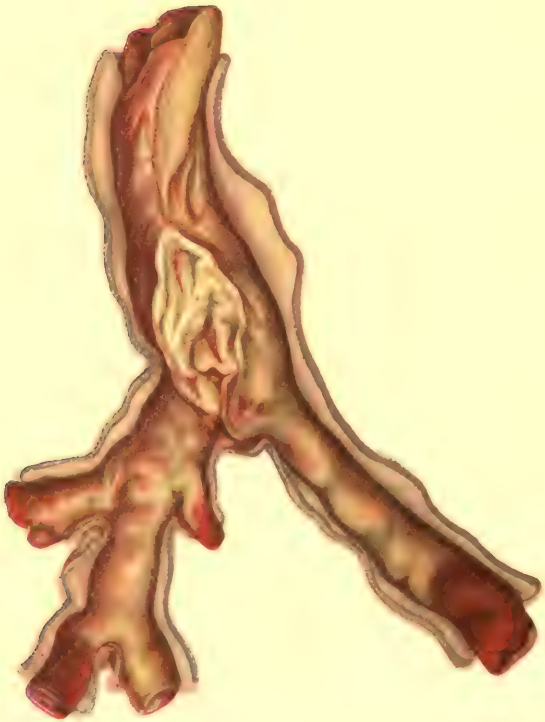
lungs. In the course of a few days further the abdomen began to swell and the bowels ceased to act. Hereupon the patient suddenly became comatose and cyanotic, the temperature ran up to 106° F., and death occurred in three hours.

But the dissemination may be so abundant as to block more than half of the vessels in the lungs. In the kidneys the fat may reach the glomeruli, and may here, according to Ribbert, excite fatty degeneration of the convoluted tubes. In the brain the globules may produce small hemorrhages, often in great number, and in the heart, in consequence of interruption of the blood supply, disseminate fatty degeneration of the musculature. As fat is not excreted by the kidneys, the final fate of the fat embolus is not known.

The interference of the *air embolus* is more abrupt. The entrance of air itself is often heard as a gurgling sound, and the obstruction to circulation may be manifested by the most extreme dyspnœa, with loss of consciousness, convulsions, and not infrequently by sudden death. Where the catastrophe is not so extreme all the symptoms may subside rapidly; and as such rapid disappearance of the signs of occlusion can occur only in connection with air, the condition may be established in this way. Moreover, the symptoms of air emboli are limited to the lungs, as vesicles which are small enough to pass the large capillaries of the lungs produce no symptoms in other organs. Little or no disturbance attends slight or slow entrance of air (Sehrwald). The sudden deaths which have happened after subcutaneous injections, as of antitoxins, are sometimes attributed to the accidental introduction of air globules into the veins. The tendency of the present time is to attribute these deaths to causes inherent in the individuals—*e. g.* to vaso-motor paralysis (heart failure).

Brain.—Occlusion of a large artery in the brain makes itself immediately manifest with abolition of consciousness, of motion and sensation, with the other signs of the apoplectic insult which may terminate in death. When the patient survives the effects of general (indirect) pressure the lesions which strictly pertain to the region of the occlusion become apparent. It is only in a rare case that embolism produces no loss of consciousness. Convulsions are not so constant. Compulsory movements show themselves, especially in embolism of the anterior portion of the thalami. There is no rule regarding disturbance of sensation, as the reflexes may be increased or diminished. There may be disturbance in the psychical sphere. Of all the organs of the body, the brain is the most sensitive to the blood supply, and abolition of function occurs immediately upon arrest of circulation. Moreover, tissue (molecular) change sets in with such rapidity that the brain cells suffer irreparable injury unless the circulation is immediately restored. In experiments made with animals and upon the heads of decapitated human beings, where soon after execution the blood was reinjected, it was found that after the lapse of as short a period as twelve minutes no centre in the brain could be excited by the blood current. Sensations and voluntary expressions were exhibited only after immediate transfusion, and then for a number of minutes. The sudden exsection of such a great vascular domain in embolism makes easily understood the symptoms of an apoplectic insult (Marchand).

PLATE VII



Marantic Thrombosis of the Uterine Veins.
(Lehmann's Atlas.)

The earliest macroscopic signs of softening of the brain were seen in ten and twenty-three hours after the experimental production of embolism in the brain.

Occlusion of a smaller artery shows the signs of abolition of function (aphasia, monoplegia, etc.) without apoplexy. The region of occlusion is marked by anæmia, hemorrhagic infarction, and later red softening, the evidence of which is visible microscopically in ten and macroscopically in twenty-four hours. Hereupon the process of yellow softening sets in.

Multiple embolism of the cortex of the brain leads to immediate syncope with loss of consciousness—signs of grave import, which disappear, however, very rapidly, to leave no trace on account of the extensive inosculation of the cortical vessels. If local symptoms of short duration disappear later elsewhere, it is for the same reason or because the affected parts were in border regions where collateral circulation could be established at once.

Whether or not chorea is caused by multiple embolization is undecided. Money produced chorea in dogs by the injection into the carotids of starch, which was arrested in the minute vessels of the cerebrum. But the symptoms from the same lesions are not always the same in man and the dog.

Pulmonary Artery.—Embolism of a chief branch of the pulmonary artery is marked by extreme anxiety, dyspnœa, cyanosis, suffocation, convulsions, syncope, and sudden death. This accident is noticed most frequently in pregnancy, parturition, and the puerperium, and in a great many cases in connection with or as a sequence to thrombosis of the femoral vein, which produces the well-known *phlegmasia alba dolens*. Sperling made a critical analysis of 33 cases recorded in the literature, finding phlegmasia in 14. The femoral thrombosis was recognized in these cases by the induration of the vein, which felt like a hard cord, as well as by the prominence of the subcutaneous venous network. The thrombotic process begins in the veins of the pelvis or at times in varices which result from compression of the great vessels by the gravid uterus. Thrombi may lie quiescent in the pelvic veins, as after puerperium and in obesity; also in cases of heart failure, to be suddenly dislodged with the most disastrous effects. Well known are the cases of women who have apparently recovered from the puerperium and sink suddenly with syncope. Bäumler reports a case of an old corpulent woman affected with emphysema and dilatation of the heart who without dropsy succumbed to repeated emboli of the lungs from thrombotic pelvic veins.

Wyder reports 12 cases of embolism of the pulmonary arteries, 8 of which were fatal. In the fatal cases an operation had been done three times for castration, twice on account of myoma, once on account of double perioöphoritis with retroflexion of the uterus. In the first of these cases the primary thrombus was situated in the great saphenous vein, marked by great swelling of the left leg. The patient had repeatedly suffered from varicose ulcers and swelling of the leg. In the other 2 cases there had probably been originally thrombosis in the domain of the pelvic veins. (See Plate VII.)

Phlegmasia is therefore always serious, in that it may entail embo-

lism of the lungs. Embolism of the pulmonary artery occurs also as the result of the so called autochthonous thrombosis of the pulmonary artery, which shows itself in convalescence from the graver infections or in disease attended with marasmus and cachexia as the immediate result of heart failure.

Embolism of the lungs occurs in general, following the main current, in the right lung, and, as determined by gravity, in the lower lobe. The left is attacked only later when the circulation in the right lung is largely blocked off. Foudroyant cases die suddenly under the picture of shock, with profound pallor and syncope, before there is time even for the development of cyanosis or asphyxia. Where the patient survives the immediate effects or when the process occurs in the course of the smaller subdivisions, the cyanosis and dyspnœa may disappear and the patient may finally recover. But in a large proportion of cases the condition is substituted later by œdema or embolic pneumonia, which often proves fatal. The development of hemorrhagic infarction of the lungs is marked by the occurrence of chills, with rapid respiration, with pain in the side, and within one to three days by the expectoration of bloody sputum. During the stage of anæmia the sputum is scanty; with the occurrence of infiltration it becomes abundant and may be profuse. The sputum now assumes a dark red or nearly black color, shows hæmatoidin crystals, but no sound or intact blood corpuscles.

Where the infarction is of any magnitude it may be recognized by physical signs, dulness on percussion, and bronchial respiration. The absence of crepitus and of the friction sound distinguishes the condition from pneumonia and pleurisy. Occlusion of a larger number of the smallest vessels will have the same effect as the occlusion of a single large branch, but occlusions of only a small number of the finer vessels will probably show only slight dyspnœa with mild rigors and difficulty of breathing, which disappear rapidly with the free establishment of collateral circulation.

Embolism of the coronary arteries shows itself with the alarming anxiety, the indescribable pains in the region of the heart, which literally transfix the patient, and which irradiate to the left arm, sometimes with cyanosis, syncope, and sudden death—symptoms which so distinctly present the picture of angina pectoris. When the patient recovers from the immediate effects the sensorium is free and the action of the heart seems perfectly normal.

Embolism of a splenic artery may reveal itself with chills, vomiting, and pain in the region of the spleen. The condition develops most frequently in connection with the lesions of endocarditis. Selter described a case in which all the branches, with the exception of one very small vessel, were blocked by emboli on the other (distal) side of an aneurysm in the splenic artery, which had developed in consequence of increased blood pressure in the course of a varicose endocarditis. Four of the cases described by Ponfick could be attributed also to increase of blood pressure.

Embolism of the mesenteric artery is marked by chilly sensations, with distention and colicky pains, and especially by the occurrence of severe enterorrhagia, with the discharge of black blood of excessively

fetid odor, the result of decomposition probably from lack of bile, as the circulation in the liver and portal vein is usually disturbed in these cases. Sometimes blood escapes internally in quantity, and accumulations which constitute real hæmatomata may be felt between the folds of the mesentery. Where the process of closure is more slow, so that collateral circulation can be established, or where it implicates only small branches, the symptoms are correspondingly slight and transitory. Tenesmus, with the discharge of fresh red blood, points to embolism of the inferior mesenteric artery.

The difficulty of diagnosis in these cases is well illustrated in the following case reported by Lochte : A laborer aged fifty-one was brought to the hospital affected with dyspnœa and cough. The examination showed emphysema of high degree, slight bronchitis, and arterio-sclerosis of the peripheric vessels. In the course of a few days the patient was suddenly seized with violent pain in the right lumbar region. Collapse set in at once. The most critical clinical investigation failed to disclose any sufficient cause for the condition. As the patient had a hernia on the right side, the idea of an internal incarceration in consequence of adhesion of the omentum was entertained. The patient died in seventeen hours. The autopsy revealed commencing necrosis and gangrene of the intestine, with total occlusion of the superior mesenteric artery by an embolus 4 cm. long. The point of origin of the embolus was found in a mural thrombus in the left auricle.

Embolism of the kidneys, which is much more frequent, is attended with pain more or less severe in the region of the kidney, and with hæmaturia and albuminuria. The symptoms may disappear in a few days and reappear later in the course of the infarction.

Embolism of the tympanum is marked by minute effusions of blood which may be visible on inspection on the membrane of the drum or may be disclosed on autopsy in the structures of the cavity.

Embolism of the retina which occludes the central artery produces sudden blindness. Occlusion of some of the smaller branches produces only local defects in the field of vision, which may subsequently disappear. The whole process of embolism may be sometimes successfully studied in the retina with the ophthalmoscope. The centre of the retina, the papilla, becomes anæmic, the empty arteries resembling white threads. The blood from collateral capillaries circulates first one way and then another (*va et vient*). The macula assumes later a dark coloration, and atrophy of the retina and papilla sets in. Sometimes the point of deposit of an embolus is visible as a small thin white scale (Sehrwald).

Ehrle reported 21 cases of total or partial embolism of the retinal arteries observed in Tübingen. The proportion of embolism to other diseases of the eye in the clinic was 1 : 3600—in private practice, 1 : 1600. Of the 21 patients, 7 had not a healthy heart, 3 had arterio-sclerosis. The embolism was total 12 times, partial 9 times. Atrophy of the optic nerve developed itself in time in one half the patients. Iridectomy and paracentesis was done without any particular result. In 1 case the sharpness of vision was markedly increased by massage.

Januszkiewicz reported 2 cases which are of great practical value, as recovery in case of puerperal sepsis, with embolism of one or both eyes, is not often observed. Both cases occurred in the clinic of Hirsch-

berg. In one case a woman, aged thirty, had high fever of several weeks' duration after an abortion. The left eye was attacked on the eighth day after the delivery, and blindness set in two days later. Metastases were found in various organs; numerous abscesses had to be opened. In the course of a few months suppuration took place in the left eye and eventuated in phthisis. The urine had always been free of albumin. The patient recovered. In the other case a woman aged thirty-six was attacked, four days after a normal labor with the seventh child, with a fever which lasted eleven weeks. A few weeks later there set in, with violent pains in the frontal region, swelling of both eyes, and as the patient tried to open them she found that she was blind in both. The eyeballs were phthisical. Nothing further was known of the later course, as the patient half a year after the delivery left for her home in Russia.

DIAGNOSIS.—The differentiation of embolism from cerebral hemorrhage is often difficult and is sometimes impossible. Embolism is more common in earlier life and in the female sex. It occurs more especially in connection with valvular lesion of the heart. Embolism is sudden in its onset; it is usually unattended with prodromata. An embolus prefers the artery of the fissure of Sylvius on the left side; embolus is, therefore, more apt to show aphasia. Dépôts in the medulla almost always depend upon embolus. Cerebral hemorrhage is more frequent in old age and in man; it is found often in connection with arterio-sclerosis, cirrhosis of the kidney, hypertrophy of the left heart without lesion of the valves. Hemorrhage is, therefore, often preceded by prodromata, and is attended with the evidence of increase of blood pressure, as shown in the comatose state, flushing of the face, throbbing of the carotids, etc. Symptoms which can be localized about the corpora striata, optic thalami, or in the pons more distinctly indicate cerebral hemorrhage.

Embolism is distinguished from thrombosis by the fact that the symptoms of embolism are sudden, while those of thrombosis are usually of slow development. The existence of heart disease speaks in favor of embolus; of arterio-sclerosis, in favor of thrombosis. The implication of different organs occurs only in embolism.

PROPHYLAXIS.—For prevention of the air embolus in placenta prævia Freudenberg suggests that after version the hand should remain in the uterine cavity. As it has been noticed that there is much liquor amnii in the cases of death, the hand is to remain to act as a tampon, allowing the water to flow away only gradually, and to excite the uterus to energetic and permanent action, so that the aspiration of air by the relaxed uterus may be prevented.

The TREATMENT of embolism is that of Thrombosis (p. 588).

PHLEBITIS.

DEFINITION.—Phlebitis (φλέψ, a vein), inflammation of the veins. The term is much more limited in our day, with the exclusion of the processes of thrombosis and embolism, which are discussed separately

(pages 580, 589). Many of the lesions and most of the dangerous symptoms formerly ascribed to phlebitis are now considered under thrombosis. Affections of the veins are less frequent and less important than similar affections of the arteries. Simple hyperplasiæ are observed more especially in the external layer, and constitute an important element in periphlebitis. Fibroid thickenings are distinguished as phlebo-scleroses. Fatty degeneration is not infrequent, but atheroma is more rare and is less extensive. Calcifications which occur in thrombi are called vein-stones or phleboliths. Processes of thickening which lead to occlusion of the vein constitute the phlebitis obliterans.

PATHOLOGICAL ANATOMY.—Pathological affections in the veins are excited, as in the arteries, chiefly by infectious processes. Weigert separated the two forms of tuberculosis, the acute miliary and the chronic tuberculosis of the wall of the vein. Virchow and Charcot described and demonstrated a syphilitic gummatous phlebitis, and the English writers refer to a special form in connection with gout as an acute gouty phlebitis. Tuberculous phlebitis is attended with infiltration and caseation, with the destruction of tissue, which may discharge bacilli into the circulation. Syphilitic phlebitis is seen most frequently in the umbilical vein of the newborn and in the branches of the portal vein.

The type of simple inflammation of the vein is represented in the phlebitis which occurs in connection with varices, and begins as a periphlebitis with infiltration (œdema) of the neighboring connective tissue.

Purulent phlebitis is the local expression of an infectious process which may be conveyed to the wall of the vein by nutrient vessels or develop in the interior of the vein from infected or suppurating thrombi and metastatic abscesses. Infection from the outside finds its best illustration in liver abscess, which if it attain the size of a walnut must somewhere reach a vein. Acute miliary tuberculosis furnishes the best example of inflammation from products carried to the walls of a vein by its own vessels.

Umbilical phlebitis may be congenital or acquired. Infectious matters are introduced more often through the open arteries than through the collapsed veins. The process usually remains local, even when it arises in consequence of puerperal infection. But it may extend to the body to develop, according to the character of the microorganisms admitted, dermatitis, erysipelas, sepsis, trismus, tetanus, or gangrene. The diagnosis of umbilical phlebitis—or rather arteritis—is sometimes made by demonstration of the *streptococcus pyogenes* in the blood or in metastases in the lungs and joints.

Pylephlebitis.—Suppurative inflammation of the portal vein is distinguished as pylephlebitis. The process begins in the radicles of the portal vein as the result usually of typhlitis (appendicitis), proctitis, prostatitis, dysentery, typhoid fever, inflammation of the umbilical vein, etc. In one case the disease developed from perforation of the stomach wall by a fish bone.

Phlebitis of the veins of the skull, *osteitis phlebitis cranii* (Bruns), is one of the manifestations of pyæmia. Affections of the facial ophthalmic and occipital veins develop as the result of alveolar periostitis, erysipelas, furunculosis, etc. of the face and neck. Phlebitis in these regions may be the cause of thrombosis of the cerebral sinuses.

Symptoms.—The symptoms of phlebitis depend upon the situation and function of the vein and the character of the inflammation. The most common expression of phlebitis, the femoral phlebitis, runs its course under the picture of thrombosis. With a more or less sudden arrest of circulation the leg becomes œdematous to such a degree that the skin is distended and shiny. Pressure leaves deep pits. The vein may be felt as a hard cord, tender to pressure, or may be seen as a blue stripe under the skin. The condition is the well known picture of *phlegmasia alba dolens*, which, for reasons cited elsewhere (page 586), is most frequently encountered in puerperal affections.

Umbilical phlebitis varies in every degree of intensity. It is sometimes a benign and comparatively localized process, but is usually attended with pain in the abdomen, fever, anorexia, uncontrollable hemorrhage, and rapidly fatal collapse. Wiederhofer pointed out in icterus, which develops in grave cases, a symptom of great importance in diagnosis. Sometimes gentle pressure on the abdomen from the symphysis up may express from the stump of the cord a few drops of pus. This evidence, which can be obtained only exceptionally, is pathognomonic of umbilical arteritis.

Pylephlebitis, purulent infection of the portal vein, is developed under the symptoms of pyæmia—to wit, with chills, profuse sweats, frequent and weak pulse, clouded sensorium, diarrhœa, and rapid collapse. Local signs on the part of the liver, pain, icterus, ascites, ectasia of collateral veins visible from the abdominal wall, hemorrhoids, and hemorrhage from the bowels, may establish the diagnosis. Abscess of the liver, which this condition most closely resembles, may be differentiated in a doubtful case by aspiration.

Prognosis is always grave, as pylephlebitis usually takes life within one to four weeks.

The treatment in all cases where it differs from that of thrombosis is wholly symptomatic.

It must never be forgotten that syphilis may be the cause of phlebitis of any of the veins, including the portal vein, and that the early and systematic use of mercury and the iodides may secure complete recovery.

VARICES.

Varices, phlebectasiæ, represent the condition of aneurysm in veins, though the dilatation is more uniform and extensive than in arteries, so that the veins assume a serpentine course and present the appearance often of masses of earth-worms. Varices develop in consequence of retardation or arrest of circulation, and are found, therefore, most frequently, as they are favored by gravity in the lower extremities, or by compression in the veins of the pelvis, broad ligaments, spermatic cord, prostate gland, and lower extremity of the rectum, where the condition is so common as to have received a separate name in hemorrhoids. Typical varices are seen as the result of senile changes upon the surface, especially of the hands in old people. Ectasia of the veins is

observed also in the establishment of collateral circulation upon the surface of the abdomen, in cirrhosis of the liver, and in the vicinity of old ulcers, especially about the so-called varicose ulcers of the leg, which result from defective nutrition and from the rupture of varicose veins. The veins in a certain section may suffer sudden dilatation in consequence of severe strain, as in lifting a heavy burden, particularly in the upper extremities (Bennett).

Aside from the influence of gravity and compression, varices develop oftenest as the result of disease of the wall of the vein, and the disease in the wall of the vein results in turn from increase of pressure in the arteries. The same causes which increase the blood pressure in the arteries—that is, which produce arterio-sclerosis—may thus affect the veins both directly and indirectly. Hence varices are observed as the result of pressure, age, syphilis, strain, alcohol, tobacco, lead-poisoning, etc.

Varices begin as star-shaped bodies upon the surface in such figures as are seen in the nose and cheeks of elderly people in acne rosacea, exciting the suspicion of drinking. The fact is, the condition here is also an affection of the veins, not of the capillaries, and the causes are those which lead to long continued spasmodic contraction of the small arteries, and often to obliteration of their lumen, with disturbance of nutrition, such as alcohol, tobacco, and gout (Rubenstein). The condition is easily recognized by mere inspection, which shows former small vessels enlarged and makes visible branches which were invisible before. The tortuous mass of venous trunks which surround the umbilicus like a mass of snakes in certain cases of cirrhosis and pylephlebitis were classically distinguished by Cruveilhier as the *caput medusæ*.

SYMPTOMS.—Among the symptoms of dilatation are certain disturbances in function and nutrition. The sensitive nerves may show paræsthesia and neuralgic pain; the secretions are altered in the mucosæ; inflammatory signs set in, proctitis (and dilatation of the hemorrhoidal veins), venous stasis in the domain of the superior vena cava. The skin may show affections of the lymph vessels, elephantiasis, as in the lower extremities and in the nose. Various eruptions may occur, eczema, furuncles, erysipelas, phlegmon, or the surface may break to form ulcers. Infectious processes may induce phlebitis with thrombosis.

A coagulum in a vein may not only cause the signs of occlusion, as œdema, but may give rise by detachment to fatal infarction in the lungs or heart, or may, in case of infection, lead to pyæmia.

The TREATMENT includes rest, the recumbent posture, elevation of the affected members, support by gentle compression, as in the leg by elastic stockings or in the scrotum by the suspensory bandage, and the prevention of rupture of the internal vessels, as of the œsophageal veins, by the relief of ascites, etc. The further treatment of varices belongs to the domain of surgery.

DISEASES OF THE MEDIASTINUM.

By IRVING S. HAYNES, M. D.

Synonyms.—German, Krankheiten des Mediastinum; French, Maladies du médiastin.

Anatomical Relations.—The mediastinum is the central space within the thorax bounded laterally by the parietal pleuræ facing the inner surfaces of the lungs; in front by the sternum; posteriorly by the vertebral column; below by the middle portion of the diaphragm; and above by the root of the neck at the level of the upper opening of the thorax. In anatomy this space is divided into the anterior, superior, middle, and posterior mediastina.

The anterior mediastinum lies in front of the pericardium, and corresponds to the area of the præcordial dulness. It is occupied by a little connective tissue and a few small lymphatic glands.

The superior mediastinum is that portion of the space above the level of the pericardium. It contains the thoracic portion of the trachea and œsophagus, the thoracic duct, the transverse arch of the aorta with the innominate, left common carotid and subclavian branches, the innominate veins, and the beginning of the superior vena cava, the phrenic, pneumogastric, cardiac, and left recurrent laryngeal nerves, the thymus gland or its fibrous remains, and lymphatic glands situated about the bifurcation of the trachea, the bronchi, and scattered throughout the space itself.

The middle mediastinum contains the pericardium, within which is the heart, the ascending aorta, and pulmonary artery, the terminations of the superior and inferior venæ cavæ. Outside of the pericardium are the phrenic nerves, termination of the azygos major vein, roots of the lungs, with their associated bronchial lymphatic glands.

The posterior mediastinum lies behind the pericardium and the roots of the lungs. Within this portion of the space are the œsophagus and the pneumogastric nerves, the descending and thoracic aortæ, the azygos veins, thoracic duct, and lymphatic glands.

All the above structures are invested and maintained in their proper positions by a matrix of connective tissue, scattered throughout which are numerous lymphatic glands (anterior, superior, and posterior groups), which, with the tracheo-bronchial glands, receive the drainage of the thoracic viscera and pass it onward to the right lymphatic or the thoracic duct.

The anterior and superior mediastinal spaces of anatomists are both included under the anterior mediastinum of clinical writers.

Inflammation of the Mediastinum.

Synonyms.—Mediastinitis; German, Mittelfellentzündung; French, Médiastinite.

Definition.—An inflammation of the lymphatic or connective tissue elements of the mediastinum. It does not include inflammation of the pleuræ bounding the mediastinal spaces at the sides, though it may result from such a form of pleuritis.

Varieties.—Inflammation of the mediastinum may be simple or infective, acute or chronic, primary or secondary.

Clinically, we have to do with inflammation resulting in the formation of fibrous tissue or pus, either diffused throughout the mediastinal space or localized by a wall of granulation tissue—mediastinal abscess. Simple acute inflammation of the mediastinal tissues, terminating in resolution, may possibly exist, but it is a condition impossible of diagnosis.

Etiology.—Those conditions which result in the production of that form of mediastinal inflammation known as the fibrous, indurated, or mediastino-pericarditis will be the ones mentioned here. The causes which usually determine the suppurative form of mediastinitis will be found under the sub-heading Abscess of the Mediastinum (page 609).

Chronic pericarditis, chronic inflammation of the pleuræ bounding the mediastinal space, chronic bronchitis, chronic inflammation of the tracheo-bronchial glands, chronic pneumonia, may result in the production of an inflammation of the mediastinal tissues of a fibrous nature.

Pathological Anatomy.—As stated, simple inflammation terminating in resolution is not capable of being diagnosed even if its existence is possible; consequently, there are no pathological changes verified by post-mortem examinations. Those causes which act steadily, but not acutely, give rise to a plastic exudation which goes on to the formation of fibrous tissue, which surrounds the bloodvessels, nerves, trachea, and bronchi in the shape of dense bands and prolongations. By the contraction of these fibrous bands as the tissue undergoes consolidation pressure effects are produced, beginning with the veins, which are least able to withstand the pressure, constricting the more strongly constructed aorta, and even narrowing the trachea or bronchi. The nerves situated in the constricted area suffer from the general pressure produced within the mass.

Symptoms.—This form of mediastinitis, usually following chronic inflammation of the pericardium, pleuræ, bronchi, or lung tissue (pneumonia), is masked by these primary conditions, and its existence may not be discovered until the autopsy is performed. If, however, in the course of the chronic diseases above enumerated there appears a fulness of the jugular veins which steadily increases; if the fulness is increased during inspiration (instead of being diminished) and lessens under expiration; if, in addition to these venous symptoms, there is a weakening of the apex beat of the heart, with disappearance of the radial pulse during inspiration, to reappear with expiration ("pulsus paradoxus" of Kussmaul),—one may be justified in concluding that the case is one of chronic mediastinitis, with the formation of dense fibrous bands sur-

rounding and compressing the veins, arteries, and other structures. Besides these symptoms there will be pain behind the sternum radiating to the side of the chest, shoulders, back, or neck, and from pressure on the phrenic or intercostal nerves, radiating to the brachial plexus. There is usually more or less dyspnœa all the time, increased on slight exertion, due to pressure on the trachea, bronchi, or produced reflexly through pressure on the pneumogastrics.

The pulse, besides being "paradoxical," is small, rapid, easily compressed, and of low tension.

A cough with mucous expectoration is apt to be present. If the case follows a chronic bronchitis or pneumonia, the cough will be modified by these diseases.

Later, cyanosis develops in the region of the neck, and may spread to the upper extremity and front of the chest.

If the return flow through the inferior vena cava is restricted, there will follow swelling of the liver, ascites, and dropsy of the lower extremities.

PHYSICAL SIGNS.—These are almost limited to an increase in the area of præcordial dulness, which may extend upward to the second cartilage, and laterally as far as the nipple line on the left, but to a less extent on the right side. The apex beat of the heart is wanting and the heart sounds are faint though regular. The pulse has been already described above as the paradoxical pulse.

In order to give a clear clinical picture of the disease the following typical case, reported by Kussmaul,[1] is quoted: A young man twenty-one years old, previously healthy, was taken ill in the latter part of October, 1872, with indefinite symptoms and without known cause. A sense of constriction and pain in the side appeared, and the patient went to bed. About the middle of December the physician found pleurisy on the left side, fever, sweats, loss of appetite, cough with watery mucous sputa, occasionally streaked with blood. During January the pleuritic exudation decreased steadily, but the dyspnœa continued; the pulse remained small, frequent, and irregular, and dropsy was observed. Later (during April) the temperature was always below normal, the pulse from 100 to 120, small and *intermitting;* it *disappeared with inspiration and returned with expiration.* The respiration was quickened. The area of pericardial dulness extended upward to the third intercostal space, passed one and a half centimetres beyond the right border of the sternum, and extended to the left nearly to the mammary line. The heart sounds were uncomplicated, muffled, and weak; their rhythm was regular, notwithstanding intermission of the arterial pulse. The veins of the neck, especially the right internal jugular, were greatly distended. The liver was swollen. The appetite was diminished, the urine was scanty and reddish-yellow, with traces of bile and albumin. In May the temperature rose above normal; the abdomen was tapped for dropsy. By June the dropsy had become very great again. The jugular veins were considerably swollen, that on the right side undulated. The distention of the veins was *increased* during inspiration. The patient grew worse, and death ensued about the last of June from

[1] *V. Ziemssen's Cyclopædia of Practice*, vol. vi. p. 649 et seq.; Kussmaul in the *Berl. klin. Wochenschr.*, 1873, No. 37.

phlegmonous inflammation of the legs following their puncture to relieve the œdema.

Autopsy.—The right lung was found attached about its root by a number of tough fibres to the pericardium and the mediastinum above. Left lung: the tongue-shaped lobe was attached by stout adhesions to the pericardium and diaphragm; the upper lobe was closely adherent to the posterior surface of the pericardium up to the point where the large vessels are given off. The pericardium was attached by strong adhesions to the diaphragm, lungs, and anterior wall of the chest, and its fibrous layer was thickened and firmly adherent to the visceral layer by recent tough fibrous membrane. At the point of origin of the large vessels the fibrous pericardium was increased in thickness. Tough bands of connective tissue, enclosing the remains of crumbling masses of fibrin, extended from the base of the heart into the cavum mediastini and accompanied the large vessels upward. These indurated bands formed loops about the arch of the aorta and the ascending portion. The arch of the aorta was thereby drawn downward and somewhat bent at the origin of the arteria innominata; the ascending portion of the aorta was compressed from before backward, so that it would scarcely admit the little finger. The trunk of the arteria pulmonalis was also compressed.

In a second case recorded by the same author[1] from the point of reflection of the pericardium at the base of the heart thick, indurated cords extended into the cavum mediastini anticum and posticum, and accompanied the large vessels, constricting and shortening them by traction.

Diagnosis.—This is difficult, if not impossible, in the beginning, as this form of indurated mediastinitis is secondary to other chronic diseases of the mediastinal contents or boundaries. When the connective tissue changes have gone far enough to form dense bands of tissue around the vessels, and these by their contraction produce a narrowing in the veins and obstruct the flow of blood from the head and neck, the jugular vessels will become distended, and their distention will become greater during inspiration and subside on expiration.

In the early stages the symptoms which should direct attention to the mediastinum are the continuation of a pain or sense of constriction behind the sternum, dyspnœa, rapid respiration, rapid and small pulse rate, the pulse disappearing during inspiration. These symptoms, continuing during or after the primary disease, would indicate that the mediastinal connective tissue was involved.

In connection with the diagnosis of inflammation of the mediastinum the following case, reported by Alfred Hudson,[2] is of interest: The title is "Case simulating Pericarditis, in which a friction sound was produced by *Emphysema of the Anterior Mediastinum.*" The patient, a boy of twelve years, was admitted to the Navan Fever Hospital on Jan. 12, 1849. He stated that for the past fortnight he had had cough and dyspnœa, and three days ago had severe vomiting and purging, accompanied by frequent shiverings and violent palpitation of the heart. On the 13th his face was livid, the eyes were glassy, the surface tem-

[1] *Loc. cit.*

[2] *Dublin Quarterly Journal of Med. Sci.*, vol. vii., Aug. to Nov., 1849, p. 241.

perature was low, the chest was rounded anteriorly, and the clavicles had a deep hollow above. Short, hard, dry cough, dyspnœa, and pain across the chest were noted. The præcordial region was dull to a large extent. The heart's impulse was strong, the sounds were clear and distinct, and accompanied by a rough crackling frottement, heard most distinctly over the base of the heart and a little to the left of the sternum, and remarkably distinct from the heart sounds. The urine was scanty, the tongue red, and the pulse hard and small. On the 14th both subclavicular regions were dull on percussion, and also over the entire left side the respiratory murmur was loud and prolonged. On the 15th great prostration was present, the heart's action was faint, there was no pulse at the wrist, body and the extremities were cold. On the 16th the patient died.

Autopsy.—The areolar tissue of the anterior mediastinum was distended with air, and it overlapped the base of the heart, so it must have been compressed by this organ during its forward impulse, thus causing the crackling sound heard during life. *Heart.* The right auricle and ventricle were distended; the auricle contained a large coagulum of dark blood; the ventricle contained a smaller quantity of dark blood and a fibrinous polypus. The valves were healthy. The left lung was congested and infiltrated with tubercles. (No statement as to the location of the leakage from the trachea, bronchi, or lung tissue is given.)

PROGNOSIS.—The prognosis is very bad. The nature of the primary condition determines to a great extent the duration of life. Chronic fibrous or suppurative pericarditis would necessarily result fatally. Chronic pleuritis, with or without a purulent exudation, could by proper treatment (free drainage for pus) be kept in subjection and the life of the patient be prolonged, but in the end the termination is fatal from the primary condition or as a result of the effects of pressure upon the mediastinal structures.

TREATMENT.—This must be directed to the primary condition. Empyema demands free drainage. Purulent pericarditis ought to receive the same treatment. Nothing can be done to prevent the formation of the fibrous tissue, and, once formed, it is bound to undergo contraction and produce pressure effects. The treatment must therefore be symptomatic and supporting.

ABSCESS OF THE MEDIASTINUM.

SYNONYMS.—German, Abscess der Mediastinum; French, Abcès du médiastin.

DEFINITION.—A circumscribed collection of pus within the mediastinal space.

ETIOLOGY.—In addition to the various causes mentioned below as resulting in the formation of a mediastinal abscess, there must be present some form of pyogenic micrococci, as the staphylococci or streptococci. The staphylococci produce the milder, circumscribed forms of suppuration; the streptococci, the severer, spreading kinds.

Those causes which have been enumerated as usually resulting in the production of the indurated form of mediastinitis may give rise to

the production of pus if the infection mentioned above is superadded. The most frequent cause of mediastinal abscess is the application of some form of traumatism to the front of the chest or root of the neck. Such trauma would include fractures of the sternum, contusions, wounds, and burns of the neck or over the sternum.

Abscess low down in the root of the neck and under the deep cervical fascia would naturally burrow downward into the mediastinum. Caries of the sternum, cartilages, dorsal or even lower cervical vertebræ may form an abscess within the mediastinum. "Catching cold" is assigned in some cases as the cause of the abscesses which resulted. This probably acts by depressing the vitality of the individual to such an extent that the inflamed mucous membrane of the respiratory tract cannot resist the onward passage of the micrococci to the neighboring tracheo-bronchial lymphatic glands.

The tracheo-bronchial glands may be the seat of suppurative, tubercular, or syphilitic inflammation, resulting by infection from the adjacent trachea or bronchi. Such involvement of these glands will in the course of time be followed by inflammation of the mediastinal glands and tissues.

Perforation of the trachea, bronchi, œsophagus (or of a diverticulum from it) by ulceration or a malignant process would result in a purulent mediastinitis. The œsophagus or a diverticulum might also be perforated by a foreign body.

Actinomycosis constitutes a source of possible infection by extension from the neck, lungs, trachea, bronchi, or tracheo-bronchial glands. Erysipelas of the face, neck, or chest, pyæmia, smallpox, scarlet fever, measles, typhoid and typhus fever, and dysentery are also possible sources of mediastinal infection. Finally, in some cases no cause can be found.

Classification.—The distribution of cases tabulated by Hare[1] is as follows: There were 50 cases of acute and chronic abscesses located in the anterior mediastinum, 12 in the posterior, 3 in the entire space, and 2 not specified. Of these cases, there were 24 between twenty and thirty years of age, 11 between thirty and forty, and 9 between ten and twenty, leaving 22 to be distributed throughout the remaining years between one and seventy. The age of the youngest was three months, of the oldest sixty-one years. The average age was thirty years. Classified as to sex, there were 58 males and 10 females.

Pathological Anatomy.—The pathological appearances are those of an abscess anywhere else in the body, modified by the nature of the surrounding parts. The abscesses may be divided into (*a*) the acute and spreading, which are attended with the symptoms of acute suppurative fever and come to some termination in a short time; and (*b*) the chronic, slowly developing, which may exist for a long time without much inconvenience or many definite symptoms. The former class of abscesses have for their cause some form of pyogenic micro-organism; the latter are usually due to the tubercle bacilli.

The abscess has a limiting layer of granulation tissue which forms to some extent a barrier against the further spread of the infection. The cavity is filled with pus. If the infection is slight, the barrier will

[1] *Mediastinal Disease*, Hobart Amory Hare, Fothergillian Essay, 1889.

become consolidated, so as to constitute a fibrous sort of sac. If, on the other hand, the infection is very severe, the wall will be imperfectly formed, and will soften under the action of the micrococci, and the abscess enlarges rapidly.

The abscess worms its way in among the various structures in the mediastinum, producing a variety of pressure symptoms. If the abscess is in the anterior mediastinum, there are no structures to be involved in its spread, but the pericardium behind and the thoracic wall in front are subject to eroding action. Should the pus be in the superior or posterior mediastinum, there may take place serious changes in the structures against and around which the pus collects, even going on to ulceration and perforation of some of these important organs.

Cold or tubercular abscesses do not spread rapidly; they have a definite limiting wall, their fluid contents is unlike true pus (Senn), and they are attended by few constitutional symptoms (aside from pressure effects). Such abscesses may become infected by the micrococci of suppuration, when a very acute, rapidly spreading abscess results, which is accompanied by marked constitutional symptoms in addition to the pressure symptoms.

Symptoms.—*General Symptoms.*—The cause of the abscess to a great extent determines the acuteness of its onset and its course, and as a result the degree of the constitutional symptoms which follow. With an acute, rapidly spreading, purulent inflammation the general symptoms will be those of acute septicæmia, such as chills, fever, sweating, foul tongue, anorexia, vomiting, and constipation, succeeded by diarrhœa. There are rapid loss of flesh, great prostration, and muscular weakness. There is usually a cough, which is spasmodic, dry, and irritating without expectoration, or accompanied with sputum that varies in character from thin to purulent and blood-stained mucus. If there has been a preceding bronchitis or pneumonia, the cough and expectoration partake of the characteristics of these affections. With a cold abscess there may be no well marked constitutional symptoms aside from anæmia, irregular chills, and slight fever, with occasional night sweats.

Local Symptoms.—These are of an indefinite character, and are apt to be referred to other parts than the real seat of the disease. They comprise a sense of fulness and constriction behind the sternum, which steadily increases until it amounts to a constant, dull, aching pain. The pain may radiate around the chest and be felt in the back, shoulders, or neck. Tenderness may be found on percussion over the sternum or in the interscapular region behind; on pressure it may be present in the suprasternal notch or above the clavicles.

Pressure Symptoms.—These vary in number and severity with the location and size of the abscess. If the abscess is evacuated, they at once disappear, partially or wholly. They are not so urgent and constant as when due to the growth of an aneurysm or a solid tumor, because the abscess has yielding walls and will insinuate itself among the various structures without compressing them to such an extent as these growths. Still, pressure effects become very urgent and cause death in many cases.

Pressure on the superior vena cava is shown by a filling of the superficial and deeper veins of the neck. With pressure on one innom-

inate these effects will be seen only on that side. Later, the upper extremity is affected, and finally, if the azygos major is pressed upon, there will be a varicosity of the superficial veins of the chest and abdomen. If the inferior vena cava is narrowed, the liver becomes enlarged; there are abdominal dropsy and œdema of the lower extremities.

Pressure upon the aorta or pulmonary artery shows itself in murmurs that coincide with the systole of the heart. Pressure upon the trachea or bronchi causes a feeling of suffocation, shortness of breath on slight exertion, rapid and shallow respiration of a whistling or wheezing character. Compression of the œsophagus results in difficult swallowing, that may increase until there is complete inability to take even fluids.

Dysphagia may be the first symptom to appear if the abscess is situated in the posterior mediastinum, but if in the anterior or superior mediastinum it will come on late if at all. Nerve pressure is manifested by pain, which varies from a mere sense of uneasiness to a severe, constant aching pain that steadily increases with the growth of the abscess. The pain may be felt behind the sternum, may simulate intercostal neuralgia, and be felt at the points of exit of the intercostal nerves—be radiated to the neck, back, or shoulders. Pressure upon the pneumogastric nerves produces paroxysmal cough, vomiting, palpitation of the heart; on the left recurrent laryngeal nerve, husky voice or complete aphonia from paralysis of one or both vocal cords, inability to cough, and noisy inspiration. Pressure on the sympathetic is indicated by an inequality in the pupils.

Physical Signs.—These also will vary with the location and size of the abscess. A deep seated abscess may produce no physical signs. If it lies in the anterior or superior mediastinum, the most constant sign is an increase in the area of præcordial dulness upward as far as the suprasternal notch, laterally even to the nipple line on the left side, but usually to a less extent on the right. If it lies in the posterior mediastinum, there may be found on deep percussion an area of dulness between the scapulæ on one or both sides of the spine and of variable vertical extent. The heart may be displaced, the direction and degree depending upon the site and size of the abscess. Its sounds will be obscured, but remain regular. The apex beat will be weakened or lost. The radial pulse at the same time may be "paradoxical."

If the abscess is a large one and situated in the anterior mediastinum, on taking a horizontal position the dulness will partially disappear (Daudé quoted by Hare).

If the abscess points forward, there will be the usual signs of swelling, redness, and heat, with fluctuation, but without pulsation.

Should the abscess open externally, air may enter and leave the cavity with every inspiration and expiration.

Terminations.—If the abscess is small and the infection slight, the process may become arrested, the pus absorbed or encapsulated, and a cheesy nodule remain. If the abscess is situated in the anterior or superior mediastinum, it may point forward through an intercostal (usually the second left) space, or even perforate the sternum and open spontaneously or be opened by the surgeon. A favorable termination can be looked for in these cases if there is free drainage afforded for the exit of the pus.

The pus may burrow downward and open through the abdominal walls anteriorly. Deeper abscesses may work downward along the spinal column, enter behind the ilio-psoas fascia, and appear below Poupart's ligament as a psoas abscess. A more usual termination is for the abscess to rupture into the trachea or bronchi when the pus is expectorated, or if present in large quantity it may drown the patient; if in smaller quantities, it may be partially expectorated, but some is inspired and septic pneumonia terminates the life of the sufferer.

If rupture occurs into the œsophagus, the fluid may be vomited or pass downward into the stomach. Perforation into a bloodvessel is rapidly fatal.

Duration.—In acute cases the duration is short; the patient either recovers after the pus has been evacuated anteriorly or speedily succumbs to the septic infection. Cold abscesses may follow the usual course and enlarge slowly, the patient living for a long time and suffering very little inconvenience, but if septic infection be added to the tubercular process, a fatal termination is speedily reached.

Diagnosis.—The diagnosis is always difficult; it is in such a locality that the subjective symptoms do not direct attention to it, nor do the physical signs appear until the abscess has acquired a considerable size.

The diagnosis must be made between aneurysm, solid tumors, cysts of the mediastinum, purulent pericarditis, malignant disease, and abscess of the root of the lungs.

The distinctive symptoms of aneurysm are expansile pulsation, the bruit, a diastolic murmur, and tracheal tugging, all of which are absent in abscess. Cysts are unaccompanied by the chills, fever, sweating, rapid loss of flesh, and excessive prostration which usually are found associated with an abscess. A diagnosis from abscess of the lung may be impossible, though in this condition there would be the evidences of a concealed abscess without the typical pressure symptoms found in mediastinal abscess.

Diagnosis between abscess and solid tumors will be given under the latter heading (p. 622).

Prognosis.—If the abscess is located in the anterior or superior mediastinum and points forward, or its nature can be determined so that a free outlet can be provided, the prognosis is fair—*i. e.* 50 per cent. will recover. Middle mediastinal abscesses were fatal in all cases reported. In 14 cases of abscess in the posterior mediastinum 2 recovered. In 1 the pus escaped through the pharynx, in the other through the œsophagus.

Treatment.—But little can be said under this heading. If the abscess points externally, it should be opened as soon as possible, and a free outlet provided for the escape of pus. If there are the symptoms of tumor, increased area of cardiac dulness with symptoms of septicæmia, and an abscess behind the sternum is suspected, a fine needle should be utilized to aid the diagnosis, and if pus is found the abscess should be opened either alongside of the sternum or else by trephining a hole through it. Rupture of the abscess into the pleural cavity demands an operation for its removal if the condition of the patient justifies interference.

The following summary of a case of mediastinal abscess, reported by Weaver, will illustrate the usual course of such affections:[1] The patient, a male, fifty-five years old, was previously perfectly healthy. First symptoms appeared six weeks before he came under observation, and were those of ordinary cold, with slight cough, but no chills. The man kept at his work for three weeks, when, becoming weaker, he gave it up. For the past three weeks he has been unable to work, but has kept about the house. For the past week he has had slight fever, and has been slowly growing weaker and more emaciated. He complains of sore throat and cough, with expectoration, scanty at first; later more abundant and accompanied by fetid breath. For the past few days the expectoration has been absent for a whole day at times, then after a severe paroxysm of coughing quite a large quantity of offensive sputum would be coughed up. Physical examination on November 21st. The patient appears much emaciated, with skin dusky, tongue coated and pale, throat negative; heart, first sounds weak, otherwise negative. Temperature 101° F. A. M., 102° F. P. M. Lungs, left negative; right, about the middle of the interscapular region is an area extending about five inches vertically, over which marked dulness is obtained on percussion. This dulness gradually gives place to resonance in all directions. At the periphery of the dull area signs of partial pulmonary consolidation are obtained on auscultation. No other abnormal signs are noted. Appearance of the sputum on standing: At the top is a frothy layer, beneath which is an opaque layer of mucus, and at the bottom is a thick yellowish green layer composed of small masses. It has an offensive odor. Microscopical examination fails to show any elastic fibres or tubercle bacilli. Abundant streptococci and a few bacilli are found by means of Gram's stain.

December 13th. Slight dulness over right upper chest is present, more marked posteriorly. Expiratory sounds are prolonged and raised in pitch and the vocal sounds are slightly increased. A few moist râles are also heard. Posteriorly is an area lying in the interscapular, infraspinous, and infrascapular regions about five inches in diameter, over the central portion of which is obtained almost flatness and over the periphery only dulness.

Over the flat area auscultation reveals sounds suggesting the presence of a cavity. The patient expectorates large quantities of purulent matter every day or two. He gradually grew weaker and died on January 15th.

Autopsy.—The right pleura contained one quart of milky fluid—offensive odor. A deposit of soft, dirty yellow lymph was found over the parietal and visceral pleuræ. The lung, which lay compressed and airless against the spinal column, was adherent behind over the third, fourth, and fifth ribs from the spine outward for five inches. Below the bifurcation of the trachea was a cavity with a blackish gray ragged lining, which communicated below with the general pleural cavity of the right side. The right lung was airless, dirty grayish red on section, and offensive. The mucous membrane in the lower part of the trachea and right bronchial tube was reddened, thickened, and rough on its surface. The left lung and pleura appeared normal. Some swollen

[1] *Chicago Clinical Review*, vol. i. p. 261.

dark bronchial and mediastinal glands were found, but none showed any evidence of caseation or suppuration.

Malignant Tumors of the Mediastinum.

New growths within the mediastinum are rare, but representatives of the several varieties have been met with. Those which have been found most frequently belong to the sarcomatous variety; next in frequency are the carcinomata. Rarer tumors are the lipomata, fibromata, enchondromata, osteomata, gummata, and teratomata. Tubercular and syphilitic enlargements of the lymphatic glands form tumors within the mediastinum. Of the cysts, hydatid and dermoid specimens have been found.

Among 95 cases reported since 1870 and tabulated in Hare's monograph,[1] are 66 sarcomata and 29 carcinomata. Of 25 cases collected by myself since the publication of Hare's book, and not included in his statistics, there are 18 sarcomatous and 7 carcinomatous growths. Thus the sarcomata are from two to three times more numerous than the carcinomata. This conclusion is at variance with that given by Hare, but the following abstract of a letter received from him in September, 1895, in reference to his monograph, will interest: "In collecting the statistics therein contained I was, of course, forced to classify the cases according to the name given to the tumor by the reporter. As you will see, many of the cases collected were reported before the time when we recognized the difference between cancer and sarcoma, and therefore a number of cases of so-called cancer were probably sarcoma. This probability is increased very greatly by the fact that the mediastinum contains almost no tissue in which a cancerous growth would be likely to appear, and, on the other hand, much tissue which could give rise to a sarcoma. Personally, I believe that while my statistics seem to prove the contrary, as a matter of fact sarcoma is the most common form of mediastinal growth." That such a conclusion is true is evident when only those cases are included which have been reported since 1870, as given above.

Among the 25 cases collected by myself there are only 3 that are secondary, and these are all sarcomata. Referring to Hare's cases occurring since 1870, and confining observation to those in which definite statements are given, it seems that among the sarcomata there are 28 primary growths in the "mediastinum," 1 in the thyroid, 6 in the thymus—a total of 35 primary cases, with only 5 secondary occurrences.

Among the carcinomata 18 were primary in the "mediastinum," 1 in the thymus—making 19 primary to 2 secondary growths.

This shows conclusively that primary growths of the sarcomatous variety are about seven times more frequent than secondary; that primary carcinomata are about nine times more numerous than secondary carcinomata, and, further, that of the secondary growths there are more sarcomata than carcinomata. This bears out what one would expect to find from a study of the structure and method of secondary implication—the sarcomata giving rise to secondary growths through the medium of the bloodvessels, and the carcinomata through the lymphatics.

[1] *Diseases of the Mediastinum*, Hobart Amory Hare, M. D., 1889.

This being the case, it appears that where there are secondary sarcomatous growths they are at a distance from the primary growth, whereas in the case of the carcinomata the secondary tumor is apt to be close to the primary one. Thus, in the sarcomatous cases one came from the thigh, another from the testicle, and in the carcinomata one came from the breast.

Location.—Taking the selected cases from Hare's tables, the distribution is as follows:

	Sarcomata.	Carcinomata.	Total.
Anterior mediastinum	35	6	41
Middle "	1	4	5
Posterior "	4	3	7
Whole "	5	7	12
Anterior and posterior	6	6	12
Anterior and middle	1	0	1
Posterior and middle	0	1	1
Total	52	27	79

The 25 cases which I have collected are thus distributed, as nearly as can be determined:

	Sarcomata.	Carcinomata.	Total.
Anterior mediastinum	16	4	20
Middle, only by extension of the growth from the anterior mediastinum.			
Posterior	1	3	4
Anterior and posterior	1	0	1
Total	18	7	25

This shows that most of the sarcomata start in the anterior mediastinum (here the so-called anterior mediastinum includes the anterior and superior mediastina of anatomists) and a majority of the carcinomata begin in the posterior space. This agrees with what we would expect to find when we keep in mind the sort of tissues from which each growth is most liable to originate.

Starting Point of the New Growth.—As nearly as can be determined among the 25 cases of tumors, 4 sarcomata began in the thymus or its fibrous remains, 9 in the lymphatic (tracheo-bronchial or mediastinal, and not specified) glands, and the remaining cases were not stated. Of the carcinomata, 1 began in the thymus gland, 1 in the cellular tissue, 1 in the bronchus, and 2 in the œsophagus.

In a general way, then, it may be said that the sarcomata begin in the glandular elements (lymphatic, thymus, or thyroid), and the carcinomata in the epithelial tissues, as found in the œsophagus, bronchus, thymus gland or its remains, or even in the connective tissue. Besides these points of origin, it is possible for the cancer to spring from the pericardium, pleura, periosteum, and lymphatic glands.

Sex and Age.

Age.	Male.	Female.	Total.	Sarcomata.	Carcinomata.
1 to 10	0	1	1	1	
10 to 20	0	0	0	0	
20 to 30	4	1	5	3	2
30 to 40	3	0	3	3	
40 to 50	5	2	7	6	1
50 to 60 and over	7	2	9	5	4
Total	19	6	25	18	7

Selected cases from Hare :

Age.	Male.	Female.	Total.	Sarcomata.	Carcinomata.
1 to 10	4	1	5	5	
10 to 20	8	4	12	9	3
20 to 30	14	4	18	9	9
30 to 40	12	4	16	11	5
40 to 50	10	3	13	7	6
50 to 60	8	1	9	6	3
Total	59	19	78	52	26

The proportion of men to women is therefore as 3 to 1.

Regarding the time when these tumors appear, it seems that more sarcomata develop during young adult life, whereas of the carcinomata more appear at a later period of life. There are many exceptions to both statements.

ETIOLOGY.—No cause can be ascertained for the development of malignant tumors in the mediastinum except in the case of secondary growths, when the infection may be implanted in the bronchial or mediastinal glands easier than in the lymphatic glands elsewhere. The thymus gland or its atrophied remains has been considered the favorite starting point for the new growths (especially sarcomata) by some writers (Letulle),[1] while this close relationship is denied by others (Steven).[2]

The truth is with both sides, as among the 25 cases of malignant tumors 5 began in the thymus gland (4 sarcomata and 1 carcinoma)—a proportion probably too high, as the list includes cases reported by Letulle.

Possibly the constant irritation of foreign particles gathered up and held by the tracheo-bronchial lymphatic glands may act as a sufficient cause to start a cell activity which ends in the development of a sarcoma. Especially is one led to this conclusion by observing how many people in whom these growths develop are in middle life, for their glands have had time to become loaded with the accumulated dirt of years—dirt inhaled into the lungs and passed onward through the mucous membrane until it becomes collected in the lymphatic glands. Cohnheim's theory of an embryonic nidus taking on activity after years of quiescence may possibly account for the beginning of some tumors. Until the cause of these neoplasms is determined for other parts of the body the etiology of mediastinal cancer will be unknown. The theory of infection may be found later to be the real cause, but this is not yet proven, and can only be mentioned as possibly constituting the true source of these growths.

PATHOLOGY.—**Sarcoma.**—Under the term sarcoma are included lympho-sarcoma, round and spindle celled sarcoma, and fibro-sarcoma.

The various terms applied to Hodgkin's disease, as lymphoma, lymphadenoma, adenia, pseudo-leucocythæmia, indicate a well defined constitutional disease, a chief manifestation of which is the universal enlargement of the lymphatic glands. The mediastinal glands do not

[1] Maurice Letulle, *Archives générales de Médicine*, December, 1890, p. 641.

[2] John Lindsay Steven, *The Glasgow Med. Journ.*, June, July, August, and September, 1891.

escape this general involvement, and become enlarged with the rest, but they do not on this account deserve to be included in a description of mediastinal tumors. Such symptoms as they might produce can be inferred from those produced by the tumors under discussion.

The structure of the sarcoma consists of a delicate stroma containing a large number of small, round, oval, or spindle shaped cells. Sometimes there is very little stroma and in parts of the growth none. Surrounding the growth there is often found an incomplete sort of fibrous capsule which becomes intimately connected with the surrounding tissues, and aids, instead if limiting, the spread of the growth.

On section of a sarcoma there escapes a creamy juice and the cut surface is soft and friable. Thin walled vessels are distributed to the growth, and they ramify in the slight amount of supporting tissue in close contact with the sarcoma cells. Hence secondary infection is chiefly accomplished by means of the bloodvessels.

Metastasis.—The lympho-sarcomata resemble the structure of the lymphatic glands from which they usually spring. Sarcomata infiltrate the structures with which they come in contact, supplanting the proper tissue of the structure with their own distinctive composition of small cells imbedded in a slight fibrous matrix. The sarcomata are peculiar in that they tend to grow in the direction of least resistance, working their way in between and around the vessels, nerves, and other structures of the mediastinum, surrounding them first and infiltrating them afterward. This peculiarity is often seen in examining a case post-mortem, when it will be seen that some of the structures included within the tumor mass preserve their identity, if not in all parts at least in some, and have not yet been invaded by the sarcoma, though they are surrounded by it.

The arteries are most resistant to the spread of the sarcomatous process. They suffer less from pressure and actual disease of their coats than the veins. The thin walls of the latter are easily compressed, and then infiltrated by the growth, which may extend into the lumen of the veins until they are entirely obliterated. Even if the sarcoma has not itself occluded the veins, its presence instigates the formation of laminated clots which will plug them. This would show that the sarcoma must necessarily alter the lining of the veins, notwithstanding statements to the contrary.

The bronchi are converted into sarcomatous tissue in whole or part, and their calibre is narrowed or even closed by the ingrowth of the sarcoma. The lung or portion of it thus cut off will collapse and produce the physical signs of such a condition.

The nerves suffer compression, but not to the extent that they do in carcinoma or aneurysm. They may be crushed off as by these growths, but they are more apt to become included within the mass and their function interfered with. They may become infiltrated by sarcomatous tissue until no trace of nerve tissue is found.

Secondary involvement of the lungs, pleuræ, pericardium, and heart takes place in some cases. The extension into the lungs is usually along the tracheo-bronchial glands and the bronchus itself. The lung tissue may also become directly affected wherever the sarcoma comes in contact with it. If the pleura or pericardium is invaded, the resulting in-

flammation is of a very acute nature, as is shown by the production of an abundant bloody exudation.

The heart muscle is last to be involved. It escapes altogether in most cases, but in some is attacked by the malignant process.

Purulent inflammations do not result from the growth of a sarcoma alone; there is needed the presence of the pyogenic micro-organisms.

Sarcomata usually begin in the anterior mediastinum, but they spread so as to occupy all the spaces, reaching into the neck, growing forward and distorting the anterior chest wall, or backward into the spinal column or downward through the diaphragm.

Carcinoma.—Under the term carcinoma are grouped all growths springing from the epithelial tissue (or its derivatives), and including the œsophagus, bronchi, and trachea. Besides originating from these usual places, such growths are stated to have sprung from any of the mediastinal tissues. The structure of the carcinoma is an alveolar arrangement of fibrous tissue, the alveoli being filled with clusters of large cells. These alveoli are really lymph spaces containing the cancer cells; hence, as a rule, this variety of tumor spreads by way of the lymphatics—*i. e.* by continuity.

Carcinomata, with a minimum growth, infiltrate the structures with which they come in contact, differing in this respect from sarcomata, which usually first surround and then infiltrate the neighboring structures. Such cancerous infiltration is attended with ulceration, and if this eats into a vessel fatal hemorrhage results.

Symptoms.—The symptoms of beginning cancerous disease of the mediastinum are indefinite, uncertain, and misleading. Growths beginning in the anterior mediastinum will manifest themselves subjectively by uneasiness, increasing to a sensation of weight and oppression behind the sternum, and attended with more or less discomfort, if not actual pain. Later the pain becomes constant, with paroxysmal exacerbations. There is a spasmodic cough, with or without expectoration. If there is expectoration, it may vary from a thin mucous sputum to a thick purulent one. There is nothing diagnostic about it.

If the growth begins in the posterior mediastinal space, a difficulty in swallowing may be the first symptom. This difficulty, at first noticed on taking solids, will increase until it may be almost impossible to swallow liquids.

The first symptoms with a growth irritating the pneumogastric nerves are indigestion, gastralgia, vomiting, and asthmatic attacks. With these symptoms there are constant, rapid loss of flesh, great muscular weakness and fatigue following slight exertion, so that the patient is forced to give up his occupation very early in the course of the disease. There is no fever at first. Later there may be chills and fever, but the fever is not typical, and is not due so much to the neoplasm itself as to the conditions induced by it. Night-sweats are usually present, and are exhausting, as they always are. The functions of the kidneys and liver are carried on as usual, unless these organs become the seat of secondary growths. There is commonly disturbances of digestion, whether the œsophagus is invaded or not, as shown by anorexia, indigestion, vomiting.

The later symptoms are due to two factors: (*a*) the pressure of the

enlarging tumor upon the mediastinal contents—a mechanical effect; and (*b*) the irritation induced by the infiltration of these structures by the sarcoma or cancer—a chemico-physiological effect.

Pressure Symptoms.—Many of the effects produced by tumors are also caused by mediastinal abscess, but at the risk of some repetition they will be discussed in order here.

Vascular Effects.—These are due to the obstruction to the return circulation in the veins and the outgoing stream in the arteries. Pressure upon the vena cava superior produces an equal filling of the veins at the root of the neck, which gradually extends upward to the face, head, and outward to the upper extremities and downward on to the chest. The superficial capillaries become varicose and the surface presents a turgid, bluish appearance. The lips and conjunctivæ are cyanotic. There may be a filling of the veins of the larynx, trachea, and œsophagus, which produces a difficulty in breathing or swallowing.

One peculiarity about the venous engorgement of the upper part of the body is illustrated by a case reported by Ayres[1]—namely, that this symptom is worse in the morning before rising, and gradually disappears to some extent after being up. The reason is apparent. Following the venous engorgement there is the exudation of serum and the formation of a hard, brawny œdema, first at the base of the neck, then extending to the head, upper extremities, and chest. Pressure upon one of the innominate veins modifies the picture by confining the symptoms to one side of the body. Pressure upon the lower part of the superior cava adds to them the dilatation of the superficial veins of the chest and abdomen—the result of obstructing the return flow from the azygos major. The inferior vena cava is affected in only a small proportion of cases when the growth involves the diaphragm and is working downward. Its narrowing is attended by ascites and œdema of the lower extremities. Pressure upon the aorta leads to an enfeeblement of the carotid and radial pulse upon one or both sides of the body, the point of pressure determining the effects.

The pulse is usually regular, small, and easily compressible, and may differ on the two sides.

Pressure upon the Air Tubes.—Narrowing of the trachea leads to difficulty in breathing, which steadily increases. The dyspnœa in some cases is so severe as to keep the patient upright and gasping for breath. Even if the breathing be easy while the person is at perfect rest, the least exertion leads to severe periods of dyspnœa and the struggle for breath wears him out.

Pressure upon a bronchus causes partial or complete disuse of that part of the lung which it supplies with air. The resulting effects will be described under the head of Physical Signs, below (p. 621).

Pressure upon the œsophagus will vary from slight narrowing to complete obliteration of the lumen of the tube; the symptoms will vary in a corresponding degree from slight attacks of dysphagia on swallowing solids to complete inability to swallow even liquids.

Nerve Pressure.—The new growths do not cause such constant nor severe symptoms of nerve pressure as are produced by aneurysm.

The nerve symptoms in sarcoma and carcinoma are often due to the

[1] *Annals of Anatomy and Surgery*, 1881.

irritation caused by the contact of the cancerous disease as much as to the pressure effects of the growth.

The nerves pressed upon are the phrenics, pneumogastrics, left recurrent laryngeal, the lowest branch of the brachial plexus, the intercostal nerves, and the sympathetic cords. The symptoms of nerve implication are indicated by an uneasy sensation increasing to pain. The pain may be severe and constant, with irregular exacerbations due to slight causes, as coughing, swallowing, etc. It may be located behind the sternum, reflected around the chest, simulating intercostal neuralgia; it may appear to be in the back of the head, in the shoulder, or it may even extend into the arm. It is apt to be paroxysmal in character even if fairly constant. Pressure upon the pneumogastric nerves may result in attacks of gastralgia, nausea, vomiting, or spasmodic attacks of difficulty in breathing, resembling asthmatic attacks. The innervation of the heart may be so interfered with that it may beat rapidly at one time (released from pneumogastric control) or very slowly at another (pneumogastric inhibition). Pressure upon the recurrent laryngeal is indicated by hoarseness, partial or complete aphonia, laryngeal croup, or œdema of the larynx. Pressure upon the lowest branch of the brachial plexus causes pain, felt at the distribution of that nerve. Pressure upon the intercostal nerves is felt at their termini, usually anteriorly, and it may be mistaken for intercostal neuralgia or pleuritic pain. Pressure upon the sympathetic cord causes variation in the pupils.

PHYSICAL SIGNS.—*Inspection.*—If the tumor develops in the anterior mediastinum, there may be a bulging forward of the first piece of the sternum or at the junction of the first with the second piece. The growth may make its appearance above the top of the sternum or clavicles. The lymphatic glands of the neck may become sufficiently enlarged to be externally visible. Motion of one side of the chest may be wanting, owing to pleuritic complications, or both sides cease to move in respiration, that function being performed by the diaphragm.

The appearance of the patient is very striking if there is hindrance to the venous return and the cancerous cachexia has developed. The swollen, turgid, and cyanotic neck, head, upper extremities, and chest afford a striking contrast to the shrunken abdomen and thin and wasted lower extremities.

Mensuration.—This may possibly show a difference in the size of the two sides of the chest, but such difference is more apt to be due to pleuritic conditions (such as adhesions or effusion) than to the mediastinal growth.

Palpation.—If the growth can be felt above the sternum or clavicle, if there is an enlargement of the lymphatic glands of the neck or axilla, or if there are found secondary nodules elsewhere in the body, these, in connection with the other symptoms, render a correct diagnosis possible. The tissues at the root of the neck may not be involved, and yet the growth may implicate them high enough up, so that there is imparted to the fingers a feeling of some deep resistance felt only on one side or more on one side than the other. The apex beat of the heart will not, in all probability, be felt, but there is conveyed to the hand a sensation of a diffused impulse in the cardiac region.

Percussion.—This often gives no sign at all, especially if the growth

is small and deeply seated. If the tumor is located in the anterior mediastinum, there is found an area of dulness of variable extent behind the sternum and extending outward from it on both sides. It merges below into the region of præcordial dulness.

If the growth be very large, percussion may give flatness over its centre, which at the margins changes to dulness. To secure this the growth must be of considerable size and close to the surface. The area of cardiac dulness is increased also by the displacement of the heart downward by the growth of the tumor.

If the tumor has extended backward so as to spread beyond the limits of the spinal column, then there may be found a dull area of an irregular round or oval outline varying in extent in the interscapular region.

Auscultation.—This is negative so far as the growth is concerned, but by reason of its pressure upon a bronchus or the trachea there follow the signs of obstruction to the passage of air to and from the lungs. Vocal resonance, respiratory sound, and normal resonance will all be modified or absent in a portion of the lung which is almost or completely deprived of its air supply. The cardiac sounds will be regular, but distant, rapid, and feeble. If the aorta is compressed, there will be a murmur coincident with the systole of the heart. Over the veins at the root of the neck will be heard a hum, due to pressure upon them.

Diagnosis.—*Aneurysm of the Aorta.*—If a tumor should be mapped out by percussion which can be imperfectly felt, and which presents no bruit and no expansile pulsation (although the tumor may rise and fall with the heart's pulsations), it is possibly not aneurysm. In aneurysm, with the exception of pain in aneurysm of the transverse aorta, the pressure effects are not so constant, so numerous, nor so severe as in malignant growths.

As evidences of malignant tumor there are the enlargement of the lymphatic glands at the root of the neck or in the axilla, the development of secondary nodules in other parts of the body, or the presence of the cancerous cachexia.

In aneurysm there is cardiac hypertrophy and a consequent increase in the distinctness of the heart's sounds, with a strong apex beat; in tumor there is indistinctness and feebleness in the heart sounds, with weakening or complete disappearance of the apex beat.

Cancerous growths infiltrate the tissues with which they come in contact, and induce effects which are not due merely to pressure, but to the irritating process of infiltration; these are wanting in aneurysm. In aneurysm there is tracheal tugging at each pulsation of the heart. A syphilitic history and atheroma of the bloodvessels favor aneurysm.

Secondary mediastinal tumor would be easy of diagnosis, as the malignant condition would be evident and the thoracic symptoms follow later.

Abscess.—In tumor, in distinction from abscess, there is lacking the history of a traumatism to the front of the chest or root of the neck, or the absence of operation or suppuration in these localities. The symptoms of septicæmia are wanting, such as chill, fever, and sweating. If purulent infection follows malignant disease, all these symptoms will be

added and the diagnosis cannot be so easily made. If the swelling is in the anterior mediastinum, there is the difference to be noted between a hard tumor and a soft, fluctuating one.

A fine exploring needle brings pus from an abscess and blood from a tumor. In an abscess pressure effects are later in appearing, fewer in number, and never so severe as in tumor. With a deep abscess the diagnosis must be made from the difference in number and severity of the pressure effects, the lack of cancerous cachexia, and the duration of the disease.

If pus is expectorated or vomited in considerable quantity, with disappearance or marked diminution of pressure symptoms, the presence of an abscess which has opened spontaneously into the trachea or œsophagus is certain.

Hydatid Cyst.—The cyst does not affect the patient's health, there are no constitutional symptoms, and the local symptoms are wanting or very mild. Evacuation of the growth spontaneously or by the knife through the front of the chest or its natural rupture into the trachea or œsophagus will show the contents to be a clear fluid.

The rupture of a dermoid cyst into the trachea has been attended with the expectoration of masses of hair (see history of a case reported later), but such an occurrence in the mediastinum is exceedingly rare.

Pericardial Effusion.—If this develops alone, it will not be confounded with malignant growths, as the characteristic shape of the præcordial dulness, the associated conditions, such as Bright's disease, rheumatism, etc., the fever, and the absence of the symptoms of malignant disease, render the diagnosis certain. But if the pericardial effusion follows the malignant growth, its presence may be overlooked, unless the examiner remembers the frequency with which the former complicates the latter condition.

Malignant growths of the lungs, phthisis, chronic pneumonia, pleuritis with or without effusion, all have their characteristic train of symptoms which distinguish them from mediastinal sarcomas and cancers. Even if any of these conditions are secondary to malignant growths in the mediastinum, there is the preceding history of wasting, dyspnœa, dysphagia, venous obstruction and aortic compression, pain, and laryngeal symptoms which indicate the mediastinum as the starting point of the disease.

The question arises, A solid tumor having been diagnosed, is it a sarcoma or carcinoma? The sarcomata are two to three times more numerous than the carcinomata. Their course is rapid. The sarcomata usually begin in the anterior mediastinum, grow to form large, bulky nodular tumors, and produce a large number of pressure effects.

The carcinomata usually spring from structures in the posterior mediastinum, grow more slowly, form rounded, smooth, small tumors, and the pressure symptoms are not so numerous as those of the sarcomata.

After all that has been said concerning the diagnosis of intra-thoracic growths; after the history has been carefully considered; after the symptoms have all been analyzed and ascribed to their proper sources, —there still remains such an element of uncertainty in the conclusion that one must be very guarded in his declaration of opinion, and espe-

cially conservative in the surgical examination of the growth. Mediastinal aneurysms have more than once been opened for abscesses, and this should be kept in mind when any exploratory measures are contemplated.

PROGNOSIS.—The prognosis is hopeless if the diagnosis is correct. A fatal termination is usual within five months from the beginning of the first symptoms.

In the scirrhous form of carcinoma death has been delayed for a year, while in the medullary variety it has occurred in the short space of nine days from the appearance of the symptoms which led the patient to seek treatment.

Patients afflicted with the sarcomatous variety of malignant growth may die in as short a time as those with carcinoma; a greater number, however, live longer than the average case of carcinoma, some surviving four, six, and even eight years.

TREATMENT.—This must be directed to making the patient as comfortable as possible, to sustaining his strength, and to mitigating the extreme suffering of his last days with a free use of opium. The pain is the most distressing and constant symptom, and must be made bearable with the milder anodynes at first, saving the opium derivatives for the last. Nourishment must be carefully looked after. All the refinements of alimentation will be needed, such as beef extracts, solid and liquid peptonized foods, and milk, eggs, scraped beef, and stimulants must be given. Everything must be done to sustain the patient's strength. If food cannot be administered by the mouth, it must be given in the proper form by the rectum.

Pleuritic and pericardial effusions should be aspirated. If there is pus within the pleural sac, it should be drained by a free outlet.

Surgical treatment directed to the growth itself is evidently out of the question. It cannot be removed by the knife. The only plan which offers any hope of alleviation and perhaps of cure is the serum-therapy treatment introduced by William B. Coley in 1892.[1] The present results, however, do not bear out the extremely favorable showing made soon after the introduction of this treatment. The sentiment of the surgical profession, at present, is reflected in the conclusions presented to the New York Surgical Society in March, 1896,[2] by Drs. Stimson, Gerster, and Curtis. They are as follows:

1. "That the danger to the patient from this treatment is great.

2. "Moreover, that the alleged successes are so few and doubtful in character that the most that can be fairly alleged for the treatment by toxins is that it may offer a very slight chance of amelioration.

3. "That valuable time has often been lost in operable cases by postponing operation for the sake of giving the method of treatment a trial.

4. "Finally, and most important, that if the method is to be resorted to at all, it should be confined to the absolutely inoperable cases."

[1] See his latest paper on "The Therapeutic Value of the Mixed Toxins of the Streptococcus of Erysipelas and Bacillus Prodigiosus in the Treatment of Inoperable Malignant Tumors, with report of 160 cases"—*The American Journal of the Medical Sciences*, Sept., 1896.

[2] Quoted in the *Medical Record* of October, 1896.

The case of mediastinal cancer is "absolutely inoperable," and a trial of the injection of the toxins might therefore be justifiable.

The final value of this treatment will have to be determined in the future after a sufficient number of cases have been observed and a certain time limit (usually five years) has elapsed without recurrence.

The cases introduced below have been chosen to illustrate the history and post-mortem findings in mediastinal sarcoma and carcinoma.

Lympho-sarcoma of the Mediastinum, involving the Apex and Root of the Left Lung, reported by J. Lindsay Steven.[1]—The patient, a butcher about thirty years of age, married, of temperate, steady habits, was treated at the Royal Infirmary during August, 1888, for pleurisy of the left side. Later trouble seemed to point to phthisis complicated with rheumatism. In October, 1889, the following condition was found: Great dyspnœa, causing the patient to lie upon the left or affected side. The face presented extreme pallor and lividity of lips, with cold drops of perspiration on the forehead; there was much œdema of the ankles and body, but it was particularly noted that as regards the head and upper extremities the dropsy was chiefly limited to the left arm and the left side of the neck and head. An enlarged gland could be felt above the left clavicle.

Examination of the chest revealed absolute dulness over the whole of the upper lobe of the left lung in front; in this region also the respiratory murmur and the vocal fremitus were both quite gone; the dulness did not extend across the middle line, so far as could be made out. In the left axillary region and at the left base posteriorly the percussion note was somewhat clearer, and in these regions some breath sounds could be quite distinctly made out. The heart's action was rapid and feeble, and the cardiac sounds were replaced by murmurs, the exact rhythm of which was not determined, but they seemed to be mainly mitral in origin.

The opinion expressed by Dr. Steven was that the case was one of mediastinal tumor which had involved the bronchi of the left lung, especially those passing to its upper lobe. His reasons for this diagnosis were the very absolute nature of the dulness and its limitation to the upper lobe of the lung, the base being left comparatively free and evidently receiving a fair supply of air; the presence of enlarged glands and painful swellings, which might fairly enough be interpreted as secondary in origin; and the presence of œdema on the left side of the neck and in the left arm.

Post-mortem a Few Days Later.—On removing the sternum a large white nodulated mass, from which, on being cut into, a white creamy juice escaped, was found occupying the upper portion of the mediastinum. This mass was in close relationship with the upper lobe of the left lung and the upper extremity of the pericardium, and the left lung was found to be very firmly adherent over its whole extent. The right lung was non-adherent and presented nothing remarkable. The liver was studded with numerous small nodules which presented the appearance of secondary growths.

Examination of the tumor showed that it was composed of greatly enlarged lymphatic glands, which had only partially remained isolated

[1] *Glasgow Med. Journ.*, June, 1891.

from one another, and the whole mass was closely related to the great vessels and bronchi. The left innominate vein passed over the front of the upper part of the tumor, and into it opened numerous veins from the tumor. The growth extended backward under the arch of the aorta, and at one point was firmly incorporated with the arterial wall, so that a distinct depression and puckering of the internal coat had thereby been produced. Still farther backward the tumor tissue became firmly adherent to the left bronchus immediately below the bifurcation, although neither the trachea itself nor the right bronchus seemed to be in the least involved. The entire wall of the left bronchus for a distance of two inches below the bifurcation of the trachea was converted into tumor tissue, so that the mucous membrane had disappeared, and the bronchus at this point was simply a channel through the growth. Below this the bronchi passing to the lower regions of the lung were found to be comparatively free and patent, but the bronchus passing to the upper lobe, which was given off in the midst of the diseased portion, was almost occluded, and during life very little air could possibly have entered it.

At its left border the mass just described was firmly adherent to the anterior margin of the upper lobe of the left lung, so that it was impossible to separate them without tearing the tissue. Inferiorly the mass was similarly adherent to the upper portion of the pericardium, and numerous nodules were found in its tissue and one or two on the wall of the heart. On cutting into the left lung its tissue in the neighborhood of the root and for a considerable distance around this was found to be converted into a white, soft, almost encephaloid structure. From the main mass of the new pulmonary tissue the tumor showed a tendency to encroach upon the lung, mainly by extending along the walls of the bronchial tubes and to a much less degree along the vascular walls.

A recent acute pericarditis with fibrinous exudation and moderate adhesions was found to involve the whole of the anterior and left regions of the pericardium, the source of irritation undoubtedly having been the advancing tumor. Several nodules of the tumor were found in the visceral pericardium on the surface of the heart, and almost the entire wall of the left auricle had been transformed into tumor tissue.

Primary Cancer of the Mediastinum, originating in the Tissue of the Right Bronchus, reported by J. Lindsay Steven.[1]—The patient was a man aged twenty-four years who was treated in the Glasgow Royal Infirmary. The symptoms from which he suffered and the physical signs in his chest led to a diagnosis of phthisis pulmonalis. He was admitted laboring under a cough and a spit of about nine weeks' duration. Expectoration was profuse, night-sweating very marked, and diarrhœa troublesome. A tumor the size of a small orange was situated on the front of the right shoulder, and another about the same size was found in the abdominal wall. Percussion over the right apex in front was quite dull, the respiratory murmur was tubular, and there were crepitant râles. The left lung seemed to be normal. Posteriorly percussion was dull all over the right lung; the respiratory murmur was tubular at the apex, and there were muco-crepitant râles at the base. There had been no hæmoptysis till the 15th of January, 1890, and then it was

[1] *Glasgow Med. Journ.*, Aug., 1891.

only slight; the sputum was nummular, the urine was normal, and the temperature was markedly hectic.

Post-mortem, February 10, 1890.—External appearances: There is a marked deformity of the chest wall. In the right lumbar region of the abdominal wall is a firm rounded tumor; and a similar smaller, but harder, rounded mass, about the size of a small orange, is situated on the front of the right shoulder. Chest: The anterior margin of the right lung is firmly adherent to the sternum. On opening the pericardium one or two moderately recent adhesions are found between the surface of the right ventricle and the parietal layer. The heart is somewhat dilated, and all its chambers are filled with clot; the muscular tissue is somewhat pale and rather soft, but otherwise the organ presents nothing abnormal. The right lung is firmly adherent over its whole surface, diaphragmatic as well as costal. It is solid from apex to base, the consolidation for the most part presenting somewhat the characters of gray hepatization, with here and there distinct nodules presenting features somewhat similar to those of caseous or catarrhal pneumonia. Surrounding the right bronchus where it runs into the lung is a hard, pearly white mass, which encroaches upon the pulmonary tissues of the root of the lung. Where this mass involves the lung its tissue presents a caseous appearance, at first regarded as caseous bronchial glands. In the anterior aspect of the upper lobe a large ragged cavity is discovered, into which projects at one point the white tissue already described as involving the root of the lung. The apex of this lung is capped by a greatly thickened œdematous pleura. The left lung is somewhat œdematous in its lower lobe; its upper lobe is much shrivelled and contracted by old fibroid change; and at the extreme apex is an old cavity about the size of a hazelnut lined with a well defined membrane and nearly full of pultaceous material of a greenish color. On removing the tumor from the shoulder and the abdominal wall the naked eye characters are found to be essentially similar to those of the mass of pearly white tissue of the right lung. The abdominal viscera are not involved.

Microscopical examination shows the tumor to be a typical cancer which originated in connection with the acinated bronchial mucous glands. The cancerous growth had nothing whatever to do with the bronchial epithelium. The primary tumor, then, in this case might be called a glandular cancer of the bronchial wall.

Other Growths within the Mediastinum.

The following histories of dermoid and hydatid cysts will be sufficient to indicate their symptoms, diagnosis, and termination:

"*Hydatid Cyst of the Anterior Mediastinum, perforating the Thoracic Wall;* remarks."—King's College Hospital, case under care of Mr. Wm. Rose. Female patient, twenty-five years old, admitted Oct. 11, 1893; born and lived most of her life in the country. She has always been healthy, with exception of an attack of bronchitis six years before, which had laid her up for a fortnight. On recovery from bronchitis she one day noticed a small lump about the size of an acorn just above the right breast. It steadily increased in size, but never gave pain or inconveni-

ence. No cough or respiratory embarrassment appeared, and she sought advice on account of the size of the mass. She appeared well nourished, but anæmic. A smooth, hemispherical swelling was to be felt to the right of the median line and over the sternal ends of the second and third ribs. Commencing about a finger's breadth from the clavicle, it extended to the lower border of the third rib below, touching the sternum internally, and measuring three inches transversely and nearly the same vertically. The skin over it was unaltered in appearance and freely movable upon the tumor. A sense of fluctuation was discernible on digital examination, and, although the swelling was freely movable, it was apparently firmly attached to the subjacent structures. It was unconnected with the breast, and the fibres of the pectoralis major seemed to be stretched over it. No impulse was noticed on coughing, and respiratory and heart sounds were normal. No swelling appeared in any other locality.

On October 16th a transverse incision about three inches long was made over the most prominent part of the tumor, skin and fascia were cut through, and fibres of the pectoralis major were separated. On attempting to isolate it from its surrounding attachments the tumor gave way and a clear colorless fluid escaped. It was now found to extend between the second and third ribs deeply into the mediastinum, leading into a cavity large enough to hold about ten ounces of fluid; the sternum and contiguous ribs were slightly eroded. The fluid was evacuated, the inner gelatinous membrane was easily removed by forceps, and the cavity sponged out with 1 : 40 carbolic acid solution, and packed with gauze impregnated with iodoform and glycerin emulsion. The superficial wound was dressed in the usual way. The next day there was a large amount of blood-stained discharge. On the sixth day the patient sneezed, and, leaning forward, noticed the dressing was bright red in color. No further hemorrhage occurred; the cavity contracted and healed. Microscopical examination showed characteristic scolices. The hemorrhage was probably due to the rupture of one of the arterial twigs which arise from the innominate.

Dermoid Cyst.—An example of dermoid cyst of the mediastinum is given in *Von Ziemssen's Cyclopædia of Medicine*,[1] reported by Cordes. The case was that of a soldier who had been previously healthy. The disease commenced with indigestion and the symptoms of an acute catarrh of the stomach, and extensive, rapidly increasing dulness developed under the sternum and in the left side of the thorax; at the end of seven weeks death occurred. A large sac was found in the anterior mediastinum, which communicated by an opening with the cavity of the thickened pericardium. On the inside of the wall, which was studded with plates of bone and cartilage, were found fibrous tumors the size of a nut or hen's egg. These were covered with well formed hairs, and upon section showed acinous glands (sebaceous glands), which were connected by their stalks to hairs. The contents as usual consisted of fatty pulp.

In two other cases reported in the same work the symptoms during life resembled those of tuberculosis, but with the presence of large quantities of hairs in the expectoration.

[1] Vol. v. p. 446.

Syphilitic growths usually begin in the covering of the sternum, cartilages, or spinal column.

Tuberculous enlargement of the tracheo-bronchial glands will lead to the involvement of the mediastinal lymphatic glands. The symptoms are similar to those produced by other solid growths in the mediastinum, and show pressure upon the veins, arteries, trachea and bronchi, œsophagus, and nerves. The symptoms are usually not so severe as those of cancerous growths, nor do they increase in severity with the rapidity that characterizes the latter. The glands are especially liable to be involved in the young, and while at first the process is a localized tuberculosis, there is great danger that if the case is left to itself general tuberculosis will develop before the twentieth year.

Rachford[1] quotes Osler and Northrup as to the frequency of tuberculosis of the tracheo-bronchial glands, and then gives the symptomatology of the disease, a summary of which is inserted below. Osler[2] says: Certainly in a very large proportion of all cases of tuberculosis in children it would appear that the first infection was in these structures —*i. e.* the tracheo-bronchial glands.

Northrup in 125 cases of tuberculosis in children found these glands tuberculous in every one.

The characteristics detailed by Rachford are—family history of tuberculosis; history of exposure to tuberculous contagion, especially in infancy and early childhood; irregularity and early appearance of the menstrual function in girls; dyspnœa and pain in the side on slight exercise; proneness to catch cold; abnormal dwarfishness; progressive failure of health; neurotic diseases, nervous irritability; hysteria; incontinence of urine without apparent local cause; dyspepsia associated with chronic diarrhœa or obstinate constipation; enlargement of the external lymphatics, accompanied by a profound anæmia not otherwise explainable. Thin, anæmic children with tuberculous family histories, having asthmatic attacks, so-called, without bronchial symptoms, and having a wheezing or whistling respiration without râles, have in all probability enlarged tracheo-bronchial glands which press sufficiently upon the trachea or bronchi to produce these symptoms.

TREATMENT.—Cysts if near the surface should be aspirated or opened and treated antiseptically; syphilitic enlargements should have specific medication.

Tuberculous conditions demand the hypophosphites, iron, cod-liver oil, special attention to diet, out-door life, and exercises tending to develop the breathing capacity.

[1] "Diagnosis of Concealed Tuberculosis," *Medical Journal*, Aug., 1895.
[2] *American Text-Book of the Diseases of Children.*

DISEASES OF THE BLOOD.

DISEASES OF THE BLOOD.

EXAMINATION OF THE BLOOD; PLETHORA; ANÆMIA.

By FREDERICK C. SHATTUCK, M. D., and RICHARD C. CABOT, M. D.

EXAMINATION OF THE BLOOD.

Importance of Blood Examination.

It is easy either to overestimate or to underestimate the clinical value of the information to be derived from blood examination, and very often mistakes are made as to the kind of help which we may reasonably expect from it.

We may not feel with Hayem[1] that "the future belongs to hæmatology;" but we need not take part in the reaction against it which seems to be taking place in Germany at the present time, merely because it is evident that there are only a few diseases in which a blood examination alone is sufficient to give us the diagnosis.

In by far the larger number of cases of disease the blood examination gives us only negative information. But this negative information is sometimes of the greatest value. Moreover, it is worth bearing in mind that the large part of the information afforded by the clinical thermometer or the examination of the urine is also negative, yet we do not on that account cease to regard the evidence they give us as a very important element in diagnosis and prognosis, and so an aid to proper treatment.

Except in diseases of the kidney or bladder and in diabetes the urine does not give us the key to the situation; but, none the less, its routine examination is a matter of course with all careful physicians. So with the blood. Except in malaria and a few other parasitic diseases, and in leucæmia, pernicious anæmia, and chlorosis, the study of blood does not give us the diagnosis ready made. But, besides the large number of cases in which a purely negative examination is valuable, we have the important side light thrown on many diseases by the presence or absence of leucocytosis, and the help given in hospital accident-rooms by the count of red cells as to the presence of an internal hemorrhage or as to the amount of any hemorrhage. The blood gives us a measure of the

[1] *Du Sang*, Paris, 1889.

degree of improvement (or the lack of it) in case of secondary anæmia, chlorosis, or debility with anæmia through weekly estimations of the corpuscles and hæmoglobin. It is of great help in cases where no history can be obtained (owing to coma or where nationality is a bar to any communication) to be able to satisfy ourselves of the presence or absence of a marked lack of red cells, or a leucocytosis, or a malarial organism. Such help as this in so many directions sets the blood examination on a par with the examination of the urine. At the present time it is a matter of routine in the medical wards of the Massachusetts General Hospital to examine the blood and urine in every case at the time of entrance, and the importance of the information gained from the two sources is, in our opinion, nearly equal and of the same kind; that is, as an element in the diagnosis or prognosis of many cases and the key to the situation in a few.

The special significance of the presence or absence of leucocytosis in the diagnosis of typhoid, pneumonia, meningitis, grippe, scarlet fever, malignant disease, tuberculosis, appendicitis and other deep seated suppurations, and in hemorrhage, will be discussed later under the head of Leucocytosis. (*Vide* page 691.)

Methods of Clinical Examination of the Blood.

No attempt will be made here to describe all the methods by which information concerning the blood can be gathered. Such processes as those by which the specific gravity,[1] alkalinity,[2] or chemical constitution[3] of the blood are ascertained are here wholly omitted, not from any lack of interest in their results as contributions to science, but because their clinical significance for diagnosis has not, as yet, been demonstrated. Nor shall we try to describe all the various serviceable ways in which any one of the processes which follow can be performed. There are many instruments by which the number of corpuscles may be estimated, and many stains for cover-glass specimens of the dried blood, but we shall here describe only such as are most generally in use and which seem to us most useful.

Confined within these limits, the clinical examination of the blood means at the present time only four processes:

1. Examination of fresh blood (with or without a warm stage);
2. Estimation of the number of red and white corpuscles;
3. Estimation of the amount of hæmoglobin.[4]
4. Examination of dried and stained specimens.

Each of these will now be described in detail.

I. Preparation and Examination of a Specimen of Fresh Blood.

Wipe the lobe of the patient's ear with a damp cloth and then rub it with a dry one. This removes gross dirt, and also makes the tissue

[1] Hammerschlag, *Zeit. f. klin. Med.*, 1892, xx. p. 444.
[2] Zuntz, *Archiv f. Anat. und Physiol.*, 1893, p. 556.
[3] V. Jaksch, *Verhandlung. d. Cong. f. Innere Med.*, 1893, 556.
[4] This gives us also a reasonably accurate idea of the specific gravity of the blood and the amount of its albuminous constituents, to both of which the amount of hæmoglobin was parallel.

hyperæmic, so that a slight puncture will draw blood. Attempts to sterilize the skin are unnecessary. Use a clean surgical needle or a sharp lancet—a sewing needle, even a sharp one, gives far more pain. The needle need not be sterile. In the several thousand counts made at the Massachusetts General Hospital since 1892 the needles have never been sterilized, and no soreness or other sign of inflammation has occurred in any case. Very likely this is due in part to the fact that in all the above cases it was the rule to press out and wipe away four or five drops before any was used for examination. This is a good rule, as it serves to get the blood flowing freely and also to wash the ear in its own blood.

The puncture is best made into the most dependent part of the lobe, the lobe being held firm by the fingers of the left hand (Fig. 35). A

FIG. 35.

Puncturing the ear.

very quick stroke gives least pain, the hand rebounding like a piano hammer.

We have repeatedly taken blood from a sleeping child without awakening it.[1] It is the mistaken tenderness which slowly presses a needle through the skin that hurts a patient. The puncture should be deep enough to make the blood flow freely and without pressure after it is once started by pressing out a few drops; if the blood squeezed out by pressure is used for examination, it is apt to be diluted with lymph pressed out of the neighboring lymph spaces, so as to render it unfit for study.

A puncture one eighth of an inch deep is sufficient if the skin is thin and the ear easily made red. In other cases one may need to go in one fourth to one third of an inch—never more. Beware of bleeders.

[1] In some hysterical patients we find great difficulty in getting blood at all. A deep puncture is needed. Cf. Jourette and Cathelineu, *Progrès médicale*, Feb. 14, 1891.

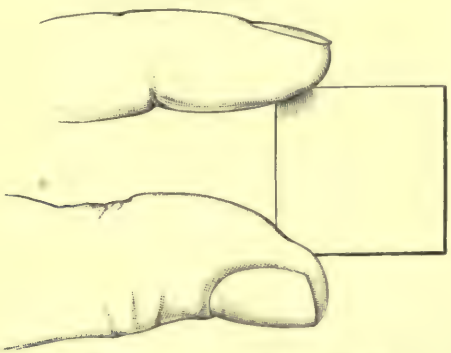
We have seen the puncture made for a blood count bleed an hour and a half before it could be checked. It is always best to inquire as to this before making the puncture, and if there is a history of hæmophilia a mere touch of the needle point will draw all the blood needed for examination without embarrassing one with a troublesome hemorrhage.

There is no doubt in our minds as to the superiority of the ear over the finger as a point for puncturing. It is far less sensitive, and a less deep puncture is needed for drawing the necessary amount of blood.

In cases of pernicious anæmia we have often found it nearly impossible to get a good-sized drop from the finger without great pressure and a deep and painful puncture, while from the ear it was obtained without difficulty. With sleeping patients it has the advantage that we do not need to rouse them to get at the ear, as we often have to do for the finger. It is also an advantage that the patient cannot watch the process.

When a drop exudes spontaneously, touch the centre of a perfectly clean cover-glass against the summit of the drop, without touching the glass to the skin, and quickly drop the cover-glass upon a clean slide, so that the force of the contact will help to spread out the blood between the two glasses. It is recommended by Thayer[1] and others to hold the

FIG. 36.



Proper method of holding a cover-glass.

cover-glasses with forceps, but we have never seen any difficulty in making preparations holding the glasses in the fingers, so long as we avoid touching their surfaces with the fingers and always hold them as in Fig. 36.

The practical importance of having both slide and cover as clean as possible is (1) that otherwise the blood will not spread itself thin enough for a satisfactory examination, and pressing on the cover-glass may distort and injure the corpuscles. (2) Dirt simulates fairly closely some of the pathological changes in the blood for which we are on the lookout, and its presence on a slide or cover leads to loss of time or to mistaken inferences.

[1] Thayer, *Boston Med. and Surg. Journal*, 1893, vol. cxxviii. p. 183.

Cover-glasses as they come from the shops are usually coated with a substance not easy to be removed by water and not always by alcohol or ether. It is safest to let them stand an hour or more in a concentrated mineral acid; rinse them off with water (without wiping), and keep them in alcohol ready to be wiped off just before using with a linen or silk handkerchief.[1] The slides, being thicker and able to stand scrubbing, are more easily cleaned, and alcohol alone is usually enough.

Where we desire to keep the blood fluid between the slide and cover for some time, a square or ring of immersion oil of the size of the cover-glass may be painted on the slide, and the cover put down so that the drop spreads out inside the ring.

Specimens so prepared will keep for hours without coagulation, and in the examination for malarial organisms this is often convenient. For clinical purposes a warm stage is rarely if ever important in the examination of the blood. The best kind is that by which the whole microscope, and not simply the slide, is put inside the warming apparatus.

Examination of Fresh Blood.—This method of examination is the best known for ascertaining the presence or absence of—

(1) The plasmodium malariæ and its after effects on the blood;[2]
(2) The filaria sanguinis hominis;[2]
(3) The spirillum of relapsing fever;[2]
(4) The rouleau formation among red cells;
(5) A necrobiotic condition of the red cells.

It is also a quick and convenient way of ascertaining with approximate accuracy—

(1) Whether any alteration in the shape or size of the red cells is present;
(2) Whether any considerable leucocytosis is present;[3]
(3) Whether the number or color of the red cells is markedly diminished;
(4) Whether the blood contains an increased amount of fibrin.

Since this very quick and simple process (a minute or two is enough for the preparation of a specimen) is capable of furnishing us with so many important data, it is one which should be as much a matter of routine as the examination of the urine, supposing that one has no time for the more exact and thorough methods to be described below.

But in order to get information from such an examination of the fresh blood we need to be familiar with the appearance of a slide of normal blood prepared in this way. Hardly a month passes without some discovery of a pathological process in the blood being recorded which the discoverer would have known to be physiological had he taken the trouble to familiarize himself with all the variety of appear-

[1] Most towels and cloths are apt to leave a scrap of lint on the glass. Thayer (*Johns Hopkins Hosp. Reports*, 1895, vol. v. p. 77) advises that the slide be rubbed vigorously by a third person just before the cover-glass is put down upon it.

[2] The description of these organisms will be found in other articles.

[3] We have known a very large leucocytosis mistaken for leucæmia by a competent observer who had examined the blood by this method only, but such mistakes would not often occur. (Page 688.)

ances which a drop of normal blood presents as it gradually dries up between slide and cover.

Probably no amount of description would enable any one to rightly interpret all these appearances. It is only by actual study of the blood itself that the student gets half consciously to disregard what is unimportant because physiological. Some of the commoner sources of error will be mentioned in connection with the directions for examining the blood in particular conditions.

Of the list given on page 639, only two need detailed description in this place—viz. I. The estimation of the amount of fibrin; II. The examination for necrobiotic conditions of the red cells.

I. **Amount of Fibrin.**—In specimens of normal blood a certain amount of the characteristic fibrin network can be seen between the corpuscles as the blood dries between slide and cover. In pneumonia, meningitis, septicæmia, and some other febrile affections the increased thickness of this network is notable, and at once strikes one familiar with the appearance of normal blood under these conditions. Its significance seems to be approximately the same as that of the leucocytosis accompanying these conditions, but no considerable stress is laid upon it by any one but Hayem[1] and his followers.

The same applies to the tendency to rouleau formation which is normally present among the red corpuscles, as seen in the fresh blood, and notably lacking in pernicious anæmia and some other conditions. It is interesting rather than clinically significant.

II. **Necrobiosis in the Red Cells.**—Maragliano and Castellino have called attention to the fact that in certain diseased conditions changes much like those accompanying coagulation (*i. e.* crenation with movements in the crenated points and of the whole corpuscle closely simulating amœboid movement, formation of vacuoles, increased affinity for basic stains, and other phenomena) may take place more rapidly than is normal. The clinical significance of such changes, however, does not appear to us to be considerable. They give little information that cannot be obtained in other ways.

Estimating the Corpuscles and Hæmoglobin.

There are two instruments useful in the estimation of the corpuscular richness of the blood, each of which has advantages of its own and to a considerable degree supplements the other:

1. The hæmocytometer of Thoma-Zeiss[2] (Figs. 37 and 39).
2. The hæmatocrit of Hedin[3] or one of its modifications, such as that of Daland[4] (Figs. 42, 43, pp. 645, 647.)

The use of each of these instruments will now be described in detail.

1. *Use of the hæmocytometer of Thoma-Zeiss.*

There are five stages or steps in this process:

(*a*) Obtaining the blood from the ear;

(*b*) Diluting and mixing the blood;

(*c*) Adjusting a drop of diluted blood in the counting chamber;

[1] *Loc. cit.*

[2] Thoma, *Virchow's Archiv*, 1882, vol. lxxxvii.

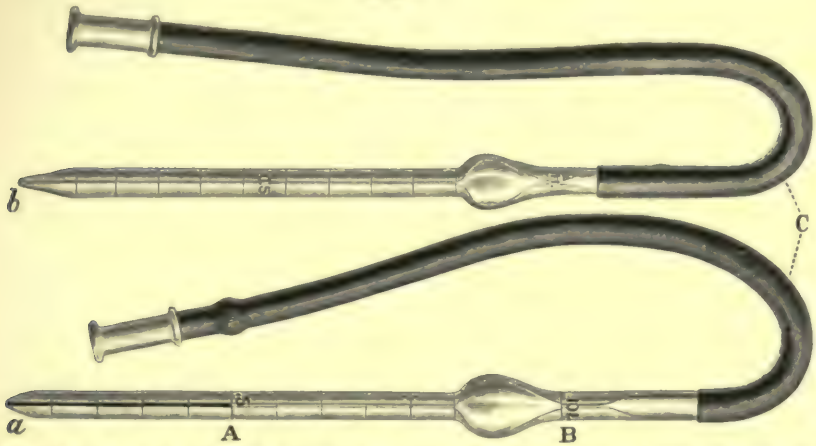
[3] Hedin, *Prag. med. Woch.*, 1891.

[4] Daland, *Transactions of College of Physicians*, Phila., May 2, 1894.

(*d*) Counting the corpuscles;
(*e*) Cleaning the pipette.

(*a*) The first step has already been described. The amount of blood needed depends on whether we are to count the red or the white corpuscles; for it is generally better to use a pipette of larger bore (Fig. 37, *b*) for the white corpuscles than for the red (Fig. 37, *a*).

FIG. 37.



Thoma-Zeiss pipettes: (*a*) small bore pipette for red corpuscles (dilution 1:200); (*b*) large bore pipette for white corpuscles (dilution 1:20). *A*, point to which blood is to be drawn up; *B*, point to which diluting solution is to be drawn; *C*, rubber suction tube.

For the large bore pipette we need the largest drop that we can make stay a moment on the ear without dropping off, while for the "red counter"[1] a small drop suffices.

When the ear has been pricked and the blood set flowing freely, the next step is the mixing and dilution of the blood.

(1) **Counting the Red Corpuscles.**—There are several solutions in use for diluting the blood in the "red counter," any of which answers the purpose excellently. We have found none better than "Gowers' solution," which consists of—

Sodii sulphatis,	gr. 104;
Acidi acetici dil.,	ʒj;
Aquæ,	ad ʒiv.—M.

With a bottle of this solution uncorked at the bedside, the point of the pipette (Fig. 38) is put into the fourth or fifth drop of blood *the instant* it emerges from the ear (spontaneously or with very gentle pressure), and by sucking gently through the rubber tube (Fig. 37, *C*) blood is drawn up to *or a little past* the point (Fig. 37, *A*) which is marked on the tube 0.5.

The easiest way, in fidgety patients, is to draw the blood a little past

[1] To save space, we shall hereafter call the small bore pipette in which the blood is diluted two hundred times, and which is used in counting the red cells, the "red counter," and the large bore pipette the "white counter."

the mark ".5," and then by tapping the point of the instrument down against a towel the column of blood may be gradually drawn down exactly to the mark. If the instrument is perfectly clean (page 645), no considerable error results from this method, and much time is saved; the aim and intention, however, should be to stop exactly at the mark. As soon as the column has been brought exactly to the mark "0.5," and the point of the pipette has been wiped dry, plunge it into the bottle of diluting solution, exert suction *the instant* the point is below the surface of the solution, and continue it until the liquid has filled the bulb and gone past it up to the point marked 101[1] (Fig. 37, *B.*) It is

FIG. 38.

Sucking blood from the ear into the Thoma-Zeiss pipette.

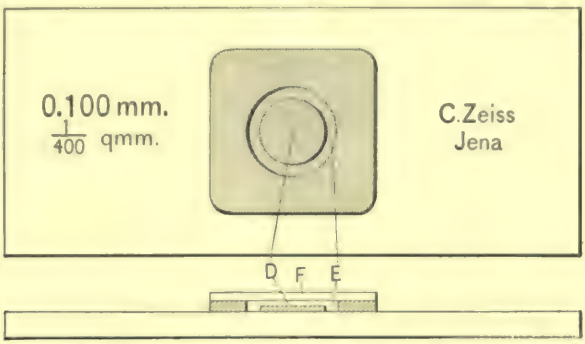
not difficult to stop exactly at the point marked 101 if the pipette is perfectly clean and dry. If not, it will be found nearly impossible, and if any mishap occurs in the process of dilution the pipette must be cleaned as directed below and the whole process begun afresh.

The blood has now been diluted with two hundred times its bulk of neutral solution, and the two are next to be thoroughly mixed by shaking and rolling the pipette, its ends being closed by the fingers. A minute's hard shaking is as good as five, as we have repeatedly satisfied ourselves experimentally.

[1] It is well to roll the pipette back and forth with the finger of the hand which holds it in the diluting solution while the latter is being sucked in. This mixes the blood instantly, and prevents any part of the drop from floating on the surface of the diluting solution, and so coming up undiluted into the narrow portion beyond the bulb, where it might possibly escape thorough mixing.

(*c*) After mixing the blood blow out five or six drops of the mixture to make sure that the portion of diluting solution last drawn into the tube (and which has not reached the bulb or mixed with the blood) is all expelled. Then blow out upon the surface of the central disk (Fig. 39, *D*) a drop of such a size that when the cover-glass (*F*) is let down upon it the whole of the raised surface (*D*) is covered, without any liquid being spilled into the "moat" (Fig. 39, *E*) around it. If this is done

FIG. 39.



Thoma-Zeiss counting slides: *D*, central ruled disk; *E*, "moat" around disk; *F*, cover-glass.

and if the whole instrument is perfectly clean, pressure on the cover-glass will bring out the concentric, rainbow-colored Newton's rings, which will remain when the pressure is removed, and the counting chamber is exactly full. To see these rings we need to get the eye near to the level of the counting chamber, so that the light from window or lamp is seen reflected from the surface of the cover-glass.

How large the drop must be can only be learned by trying, and the process must be repeated until the above conditions are fulfilled. Otherwise the count will be valueless. It is best to let down the cover-glass with a needle, as in mounting microscopic specimens, and to lose *as little time as possible* in doing so after the drop is in position. Until the concentric rainbow colored rings are seen between slide and cover no counting should begin.

(*d*) After waiting a minute or two for the corpuscles to settle thoroughly upon the ruled surface (Fig. 39, *D*), the count is begun, using preferably an objective 5 of Leitz or *D* of Zeiss and an eyepiece No. 1 or 2.

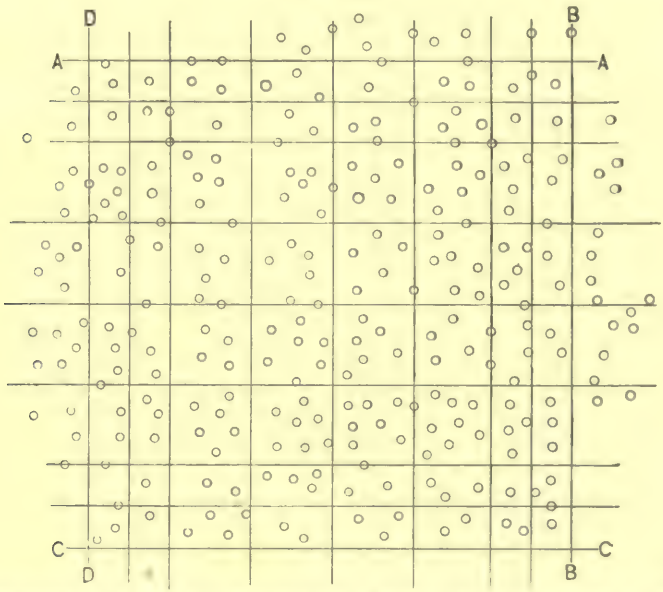
The ruled surface of the counter is divided into four hundred squares. Now, the more squares the observer counts, the less his chance of error, but some standard number must be determined upon, and the limit of error for that number allowed for in inferences drawn from the count.[1] In our experience it has seemed a good rule to count the corpuscles in five fields of thirty-six squares each, taken in various parts of the ruled space, and then repeat the process with a second drop from the pipette. If the count of the second drop differs widely from the first, a third drop is counted, and the average of all three considered the true count. If the second drop does not show results widely different from those of

[1] See Reinert, *Zählung der Blutkörperchen*, Leipsig, 1891, pp. 48 *et seq.*

the first, the average of these two is considered the true count. Thus the corpuscles in either three hundred and sixty or five hundred and forty squares are counted in all. With this number the error is not over 2 or 3 per cent. for any one who has had some practice in counting blood.

Among the difficulties met with in counting is the presence of corpuscles on or touching the outer boundary lines of the space to be counted. In counting, for example, a field of thirty-six squares (Fig. 40) we find corpuscles on the lines *AA*, *BB*, etc., and are puzzled

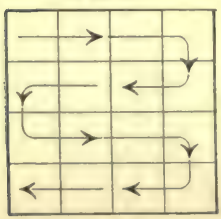
FIG. 40.



Field of 36 squares of a Thoma-Zeiss counting slide, as seen under the microscope. The number of corpuscles indicated in the plate corresponds to 5,000,000 per c.mm. (normal blood). The blood is here diluted 200 times.

whether to count them or not. To obviate this, it is well to make it a rule to count all cells on or touching two of the outer boundary lines (*e. g. AA* and *BB*), and none at all on the other two outer boundary lines (*CC* and *DD*). Of course all cells within these outer boundaries are to be counted whatever their position. Beyond this, the details of counting are best settled by each observer as he finds most accurate and simplest for himself. Our own practice is to count the squares in the order indicated by the course of the serpentine arrows in the accompanying figure (Fig. 41).

FIG. 41.



The course of the arrow illustrates the order in which the squares may be passed over in counting corpuscles.

When the number of corpuscles in three hundred and sixty or five hundred and forty squares has been estimated, that number is divided by the number of squares counted, and the result multiplied by 800,000. This gives the number of red cells in a cubic millimetre of blood.

The counting can be done either by natural or artificial light, and if our diluting solution is fresh and free from sediment and the counting chamber clean, there will be no difficulty in judging how many cells each square contains, and no need of distinguishing extraneous fragments from corpuscles.

(*e*) The importance of cleaning the pipette *as soon as the counting is done* is so great that we have made it one of the regular steps of every count. First water, then alcohol, and lastly ether, must be sucked into the bulb in succession and brought into contact with every part of the pipette. After this air must be sucked or pumped through it until it is perfectly dry, as is shown by the free movement of the glass ball within the bulb of the pipette. A hard-rubber syringe with a flexible tube fitting on to the end of the pipette is useful for this purpose. If these precautions (which take but two or three minutes) are omitted and the blood dries in the pipette, it may be a matter of hours to get it clean; and if it is not perfectly dried after cleaning, the mixing of the blood on the next occasion cannot be accurately performed.

All the steps of the process should be performed as quickly as possible until we have finished the mixing, but it is not necessary to count the blood as soon as it is mixed. The experiments of Reinert,[1] which we have repeatedly verified, show that the diluted blood may remain twelve hours (and presumably longer) in the pipette and no change result in the count. It is not necessary, therefore, to carry a microscope to the bedside of the patient. The diluted blood may be carried home (care being taken to cover both ends of the pipette by stretching a rubber band around them) and counted at leisure (after thoroughly shaking the pipette). The bulk of the hæmocytometer and a small bottle of Gowers' solution is trifling.

The normal number of red corpuscles has been usually stated as 5,000,000 to the cubic millimetre for men, and about a half million less for women. There is a tendency of late years to put these figures higher. Our experience is entirely in accord with that of Thayer and others that 5,500,000 is nearer the normal number for American men, and that 6,000,000 or 7,000,000 are not at all rare in health.

(2) **Counting the White Corpuscles.**—To make a reasonably accurate count of white corpuscles, using the pipette described above, we must either count a far larger number of squares than was necessary for the red (at least ten times the whole ruled space) or else use the "white counter" (Fig. 37, *b*). With this the technique is the same as that above described, except that instead of Gowers' solution a $\frac{1}{3}$ of 1 per cent. solution of glacial acetic acid in water is used to dilute the blood.[2] This fluid makes the red cells invisible, while the white stand out prominently and are easy to count.

The only objections to this method are that it involves the expense of another pipette; that it needs a more painful puncture and considerably more blood; and that the technique of mixing is somewhat more difficult, owing to the larger bore of the tube, which makes it hard to stop

[1] *Zahlung der Blutkörperchen*, 1891.

[2] After sucking the blood into the pipette the latter must be kept as nearly horizontal as possible during the subsequent manipulations, as, owing to its larger bore, the blood easily runs out of it, as it will not do from the red counter.

at the mark 11 and to put a drop of the right size into the counting chambers. To avoid these difficulties there are several devices by which the white corpuscles may be counted with the red counter. These all depend upon the fact that the drop of diluted blood when adjusted in the counting chamber covers a considerable area outside the space which is ruled off. Here the corpuscles settle as evenly as elsewhere, and may be utilized by the following methods, in all of which the object is to avoid the delay of putting several successive drops into the counting chamber.

One way is to measure the field of the objective used[1] and count as many fields outside the ruled space as will make up a surface equal to that of the ruled space or to some convenient fraction of it. For example: our Leitz No. 5 objective, with a No. 2 ocular, has a field almost exactly one fourth the size of the whole ruled space, so that in counting the white cells in four fields, taken anywhere outside the ruled space, we have covered a space equivalent to the whole four hundred squares. We have used this method in several hundred cases, and always found it accurate, so far as could be judged by comparing the number of cells found in many successive series of four fields each, using always the lens mentioned above.

Franklin White of the Massachusetts General Hospital shields the field of his No. 1 eyepiece with a bit of black cardboard put inside the eyepiece, and leaving a square aperture of such size that a field of exactly one hundred squares of a Thoma-Zeiss counter are to be seen through it. Four fields of a lens so shielded taken anywhere outside of the ruled space are exactly equivalent to the whole four hundred squares ruled off. This is perhaps as cheap and simple a method as any.

It has been suggested by various observers to have the whole surface of the disk *D* (Fig. 39) outside the original ruled space marked off in squares of one square millimetre each. Counting the white cells in one of these larger squares would be equivalent to counting through the whole four hundred originally ruled. I have been hitherto unable to hear of any one in this country who can do this ruling. It is necessary in any case, whatever method is used, to have at least 100–150 leucocytes as a base of computation; otherwise the chances of error are too great. Whatever number is obtained is divided, as before, by the number of squares counted and multiplied by 800,000. The normal number of white cells averages 7500 to a cubic millimetre, but 5000 to 10,000 are within normal limits, the ratio to the red cells varying between 1 : 500 and 1 : 1000.

If we count the white cells with the "red counter," using some of these devices, we have to learn by practice the peculiar brilliant refraction of the white cell, which is the chief mark of difference from the red. This is best brought out by drawing up the lens a little so that the cells are slightly out of focus. Toison's diluting solution[2] stains the

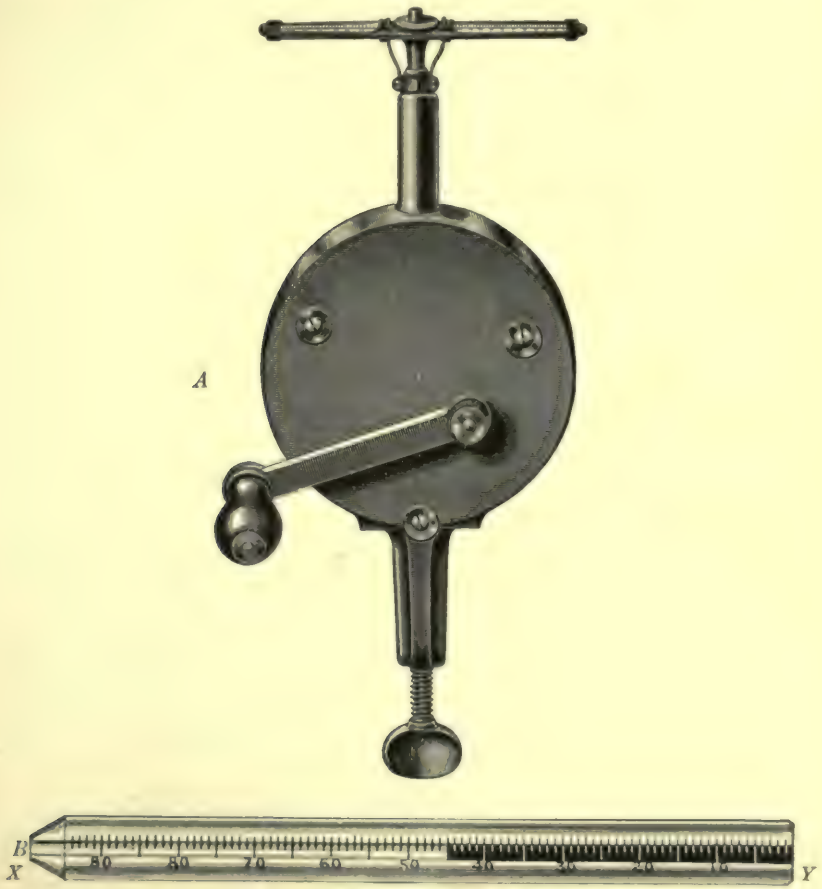
[1] This may be done roughly by simply focussing the lens on the ruled surface of the counting chamber and counting the number of squares which occupy the field of the lens.

[2] Sodii chloridi, 1;
Sodii sulphatis, 8;
Glycerini puri (neutral at 30° C.), 30;
Aquæ, 160;
Methyl violet 5 B (Merck or Grubler), 0.025.

white cells faintly violet, and is useful in this respect, but makes the subsequent cleaning of the pipette somewhat troublesome and involves a delay of ten to fifteen minutes before the stain is visible in the leucocytes.

Daland's Hæmatocrit.—The hæmatocrit of Hedin, as modified by Daland (see Fig. 42), is a comparatively new instrument, the chief

FIG. 42.



Daland's hematocrit: *A*, whole instruments with two capillary tubes in place on the horizontal whirling beam; *B*, capillary tube, showing a column of blood cells packed down at the end; *X*, end into which blood is sucked; *Y*, end to which rubber sucking tube is attached.

merit of which is its great saving of time and eyesight, particularly in those cases which are most tedious when counted with the Thoma-Zeiss instrument. According to Daland, its results are as accurate as those of the hæmocytometer except in grave anæmias and leucæmia. (On this point see below, p. 647.)

The estimation of corpuscles depends on the length of the column of cells which are packed down by centrifugal force at the end of a capillary tube filled with blood and whirled with great rapidity in a

horizontal plane. After a certain number of revolutions (the number varying with different machines and operators) the column of red corpuscles at the end of the tube is packed so tight that further whirling has no effect on it.

With Gärtner's[1] modification of the Hedin instrument the blood is diluted one half with bichromate of potash (2½ per cent.) or Müller's fluid. In Daland's modification of the instrument no diluting fluid is used, and the tube is graduated in such a way that each division of the scale (Fig. 42, *B*) corresponds roughly to 100,000 corpuscles per cubic millimetre. As before stated, this instrument is applicable chiefly to those cases where the corpuscles are *not* greatly deformed (as in pernicious anæmia or grave secondary anæmia), and the white cells cannot be satisfactorily counted with it at all. The tedium of counting cases which have 5,000,000 or more corpuscles to the cubic millimetre is greatly diminished by using this instrument, with which a count is usually done in two minutes.

The drop of blood needed is rather larger than with the Thoma-Zeiss instrument, but not larger than can be easily obtained in the cases to which the instrument is applicable. One of the difficulties with the instrument in its present form (*i. e.* Daland's modification of it) is the amount of noise it makes when screwed on to all ordinary tables. Edes, who uses the instrument among patients especially apt to be annoyed by such a noise, writes that he holds the instrument in his hand while using it at the bedside. Fear of the blood's coagulating prevents our having the instrument at any considerable distance from the patient.

Another difficulty is that, in our experience, the column of blood when packed down does not end squarely and distinctly at one of the ruled divisions of the tube, but has a bevelled end or frays off, so that it is hard to decide where to say it stops.

To use the instrument we prick the ear as above described, and, fitting a bit of rubber tubing on to the end *Y* of one of the ruled capillary tubes, suck it entirely full of blood. Then, carefully closing the free end, *X* (Fig. 42) with the finger (which should be greased with a little vaseline to prevent the blood's adhering to it), pull off the rubber and adjust the tube in the place prepared for it on one of the horizontal arms of the hæmatocrit (Fig. 43, *P*). Another similar tube should be put (empty) on the other arm of the machine to make the balance true.

All this is to be done as fast as is compatible with accuracy; the handle of the machine is then to be revolved at least seventy times a minute for a minute and a half or two minutes. Great care should be taken that the horizontal beam of the instrument is joined in such a way that it cannot possibly fly off, as such an accident might be very dangerous.

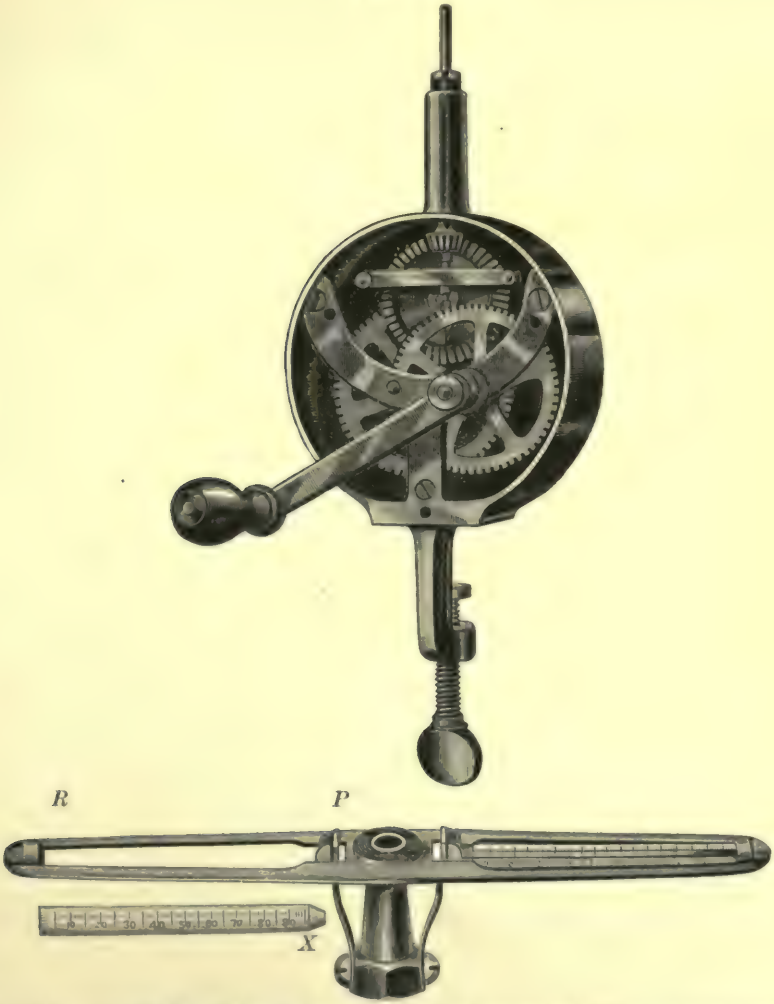
It is well to put a little vaseline at the point (*R*, Fig. 43) against which the column of blood is packed down, in order that the blood may not adhere to the metal when the tube is removed to be read off. The reading of the instrument is best done by putting the tube upon a bit of white paper. The difficulties of deciding where the column ends have been alluded to above, and are increased by the presence of the filmy, nearly

[1] Gärtner, *Berl. klin. Woch.*, 1893, No. 4.

colorless band of white cells which arrange themselves at the end of the column of red cells, but which are too vaguely seen to be capable of accurate estimation.

Each of the one hundred sections into which the tubes are marked off corresponds roughly to 100,000 corpuscles.

FIG. 43.



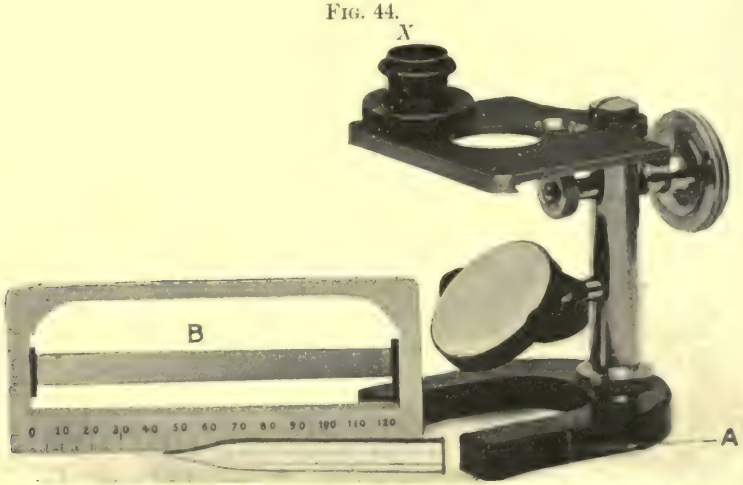
Daland's hæmatocrit; *P*, end of spring against which the pointed end (*X*) of the tube is put; *R*, end against which the column of cells is packed by whirling.

As has been said, the instrument saves a great deal of time and labor, and is claimed to be as accurate as the Thoma-Zeiss counter in cases where the corpuscles are not deformed. Where small corpuscles are present, several of which can be packed into the space occupied by one normal corpuscle, the instrument is obviously inaccurate. Further, as it has been shown by Herz and others that the volume of the indi-

vidual cell varies a great deal in many diseased conditions, even where marked deformities in size and shape are not demonstrable by other methods, it is unlikely that accuracy can be achieved by the instrument as a corpuscle-counter. It gives the volume, but inferences from this as to the number of corpuscles cannot be reliable.

Hæmoglobin Estimation.—The instrument most generally used is that of v. Fleischl,[1] which depends upon the principle of directly comparing the tint of a drop of blood after dilution with the tint of various portions of a strip of glass (*B*) whose color shades from a decided red at one end to a clear glass at the other (Fig. 44).

Fig. 44.



Fleischl's hæmatometer: *A*, capillary tube; *B*, strip of tinted glass; *X*, mixing cell.

To use this instrument, fill both sides of the cell (*X*) about one fourth full of distilled water and carry it to the bedside with the measuring tube (*A*) and a needle or lancet to be used for puncturing. The measuring tube must be carefully cleaned and dried (page 645) before using. When the drop has been made to flow freely from the ear as above described put the end of the little tube horizontally into the side of the drop, which will at once fill it if the tube be clean. After carefully and quickly wiping away any blood that may be on the outside of the little tube,[2] and making sure that the blood in it is just flush with the surface of each end, and does not present a convex or concave surface, immerse it in the water of either one of the divisions of the cell (*X*), and rattle it quickly back and forth, so that the water may be forced in, first at one end of the little tube and then at the other.

So much of the operation should be done at the bedside, and as quickly as possible, to prevent any coagulation of the blood in the tube, which in some cases takes place very rapidly. After this the cell, with

[1] In this country and Germany the great majority of observers use this instrument. In France, Hayem rules supreme in the matter of instruments, as in everything else that concerns the blood; in England the Gowers instruments are used to a certain extent.

[2] It is well to grease the outside of the little measuring tube to prevent the blood from adhering there.

the little tube still immersed in one of its divisions, may be put in place on the main body of the instrument and carried to a room or closet where daylight can be excluded and only artificial light (preferably gas, oil, or candlelight) used.

Next, the expulsion of the blood from the little tube is to be finished by forcing a few drops of water with a medicine-dropper through the tube and into the compartment where the mixing has been begun. Then, using the metal end of the measuring tube, mix the blood and water thoroughly in all parts of the compartment. After this both compartments of the cell are filled to the brim with water, care being taken that neither compartment shall overflow into the other. The compartment containing the clear water is to be adjusted so as to come over the slip of colored glass, while through that containing the blood and water the light thrown up by the mirror below will pass directly

FIG. 45.

Position of observer during the use of v. Fleischl's hæmometer. A tube of paper has been slipped over the mixing cell. Note position of candle.

to the eye. By turning the screw the glass slip can be moved back and forth until the color is the same in both sides of the cell.

It is easier to judge of the color if a sheet of paper is rolled up and used as a hydroscope by putting the end of it down over the cell and looking through the other end with one eye. It is best not to stand facing the light. We wish to avoid having the image of the one compartment fall on the upper half of the retina and the other on the lower, for the upper part of the retina is less sensitive than the lower. There-

fore we should stand or sit at the side of the instrument, so that a line dividing the two compartments runs between the observer's eyes and not across them (Fig. 45).

When the colors in the two compartments approximately match, it is well to turn the screw back and forth with short, quick turns rather than slowly and gradually, as we can judge of slight color differences better when the darker or lighter portions of glass are brought into comparison with the blood tint suddenly, and not by the gradual increase and decrease produced by slow turning of the screw.

Suppose, for example, we have decided that the color of the blood corresponds to a color of the glass somewhere between the Nos. 40 and 60. It is well to move the screw suddenly from 40 to 55, which very likely will convince us that the color at 55 is too dark; then from 55 suddenly back to 45, which may show us that 45 is too light, when, had we moved it slowly from 55 to 45, we might have been unable to decide on any one point between the two at which to stop.

Keeping up the quick turns of the screw for shorter and shorter distances back and forth, we can usually bring it down to a matter of doubt between (*e. g.*) 40 per cent. and 45 per cent. Resting the eye very frequently and trusting the impression of the first glance, rather than of a long look, enables us to read it more accurately, but there are many who can never learn to exclude an error of 5 per cent. A certain amount of error seems to us inevitable, since the portion of colored glass which appears before the eye at any one time through the aperture of the instrument includes a range of twenty degrees of color, and is not evenly colored like the diluted blood. We have, therefore, to pick out as well as we can the color of the *centre* of the portion of glass appearing at the aperture, which fades of course imperceptibly into the darker and lighter colored portions on either side of it, so that picking at the exact centre of it and comparing that with the blood tint is an impossibility of the same kind as arresting and holding fast the present moment.

But the impossibility of an exact reading does not affect the real value of the instrument. It can hardly be essential for diagnosis or treatment to know the amount of hæmoglobin within, say, 5 per cent.

It is always best to use as little light as possible in reading off the hæmogloblin percentages. The eye can judge more accurately if not over-stimulated by an excess of light thrown up through the instrument, and with low percentages the light should be turned down lower than for higher percentages. With percentages under 25 two pipettesful should be taken and the result divided by 2.

Examination of Fixed and Stained Blood Films.—The preparation of dried and stained specimens is a process which yields, on the whole, more information than any one of the other three previously described. It gives us an approximate idea of the number of the red and white corpuscles and of the hæmogloblin percentage. It is by far the best method for studying the variations in the size and shape of individual cells, and it is the only method of distinguishing the several varieties of leucocytes from each other, and ascertaining the presence or absence of nucleated red corpuscles and of degenerative and adventitious forms both of the white and the red cells. Bacteria and animal

parasites may also be studied by this method, although for the latter the examination of the fresh blood is in many ways preferable.

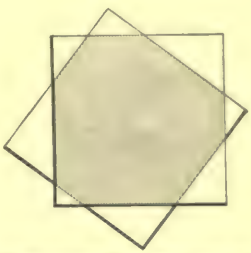
Out of the many methods used for preparing and staining specimens only those most generally in use will be described here:

I. The first and most difficult step is to spread a sufficiently thin and even film of blood upon a cover-glass. The success of the whole process depends very largely on this first step. If the blood is spread too thickly or if the shape and properties of the cells are changed by rough handling of the cover-glasses during the spreading, but little can be made out of the specimen after staining.

In the first place, it is absolutely essential to have the cover-glasses as clean as strong nitric acid, followed by water and then by alcohol, can make them (p. 639). Very thin cover-glasses serve best. At the last moment the surfaces of the glass should be dusted off with a camel's-hair pencil, as a speck of dust will spoil the preparation. Care must, of course, be taken not to touch the surfaces of the cover-glass with the fingers, but to hold it always in the way shown in Fig. 36, page 638.

When the ear has been cleaned and pricked as above described, touch the centre of the cover-glass to the tip of the drop the instant it exudes, so as to get a very small drop on the glass. Then with as little delay as possible drop this glass with the blood side upon a second cover-glass in such a way that the corners of one cover shall *not* correspond with those of the other (Fig. 46).

FIG. 46.



Illustrating the position of cover-glass during the spreading of blood films.

If the glasses have been properly cleaned, the drop of blood will fly out over their surfaces and cover the whole the instant that they touch. Then, before they have time to stick, they must be rapidly slid apart, moving them exactly in the plane of their surfaces, and not separating them. If this is done before the blood has spread over the surface of the glass, the layer will be too thick, while if we delay a second *after* the blood has spread the glasses will stick. It takes a little practice to get just the right moment for sliding the glasses apart, but if they are really clean there is no real difficulty in making good specimens. The glasses are to be dried in the air or over a flame,[1] and can then be kept for an indefinite period before staining.

II. Before staining, however, we must fix the corpuscles upon the glass, preferably by heating them. The surface of a strip of sheet copper a foot long and two or three inches wide supported over a gas, oil, or alcohol flame gets after a few minutes to be of a certain fixed temperature at any particular distance from the flame. By dropping water on the heated copper the boiling point may be easily found and marked with a line. On or just inside this line the cover-glasses are to be put *face* downward. As to the length of time that it is best to heat them observers differ very much. Thayer recommends an hour or more, but we have never been able to get any better results with an hour's

[1] This is especially useful in pernicious anæmia. The glass must be held in the fingers.

heating than with ten to fifteen minutes', and some of the best specimens have been heated only two minutes. Certainly all information of clinical importance can be obtained with fifteen minutes' heating at the boiling point of water if we use the stain described below. There is much greater danger of overheating than of underheating. An overheated specimen is so pale as to be worthless, while an underheated one, although too dark, often shows the most important points well enough.

III. Of the various stains in use, the simplest and most useful for the general practitioner is that made by mixing Biondi powder (which contains methylene green, acid fuchsine, and orange *g*, and can be obtained in any of the large cities) with alcohol and water in the following proportions:

Ehrlich-Biondi's powder,	gr. x;
Alcohol,	1 c.c.;
Water,	6 c.c.

It must be filtered before using, and sometimes requires refiltering.

Ehrlich's latest formula is as follows:

Saturated aqueous solution orange *g*,	120–135;
" " " acid fuchsine,	80–165;
" " " methylene green,	125;
Water,	300;
Alcohol,	200;
Glycerin,	100.

With a glass rod a drop of one of these mixtures is spread over the surface of the cover-glass and washed off again at the end of a period varying with the length of time that the specimen has been heated, and also with the particular characteristics of the stain made. Specimens heated fifteen minutes need about two to ten minutes' staining, using the stain above described. Specimens heated two minutes need about thirty seconds to two minutes. The other periods can be reckoned from these figures, which are, of course, only approximate.

After washing in water and drying with blotting paper they are ready for mounting in balsam and for examination. An oil immersion lens ($\frac{1}{12}$ of Leitz or its equivalent), with a No. 2 or 3 eyepiece, is most convenient for examining stained specimens, and a movable stage, although by no means essential, is very helpful in making differential counts of the leucocytes.

A cover-glass preparation of normal blood heated and stained as above directed shows the following characteristics:

Appearances of Normal Red Cells.—The red corpuscles stain *yellow*, tending toward brown or pink if underheated or overstained, while if overheated or understained the yellow is fainter. The centres are lighter than the periphery, owing of course to the bicavity. Their diameter varies between 6μ and 8.5μ—about 7.5μ on the average. If the blood has been properly spread on the cover-glass, the cells should not overlie each other nor show any crenations or deformities, unless possibly at the edges of the slides.

In most cases no conclusions should be drawn from the appearance

of cells near the margins of the slides, as it is there that damage is apt to occur in the process of spreading the blood. A certain amount of variation in color may occur even in normal and successfully heated and stained red cells, more or less of a purplish or brownish tinge replacing the clear yellow. The degree of physiological or accidental variations must be learned by practice, and distinguished from the decidedly purple or brown tints characteristic of the polychromatophilic forms.

LEUCOCYTES.—Ehrlich's triple stain brings out more clearly than any other known method the characteristic points of the different varieties of white cells in the blood. The amœboid movements and phagocytotic faculties of certain kinds among the leucocytes are best seen in the fresh blood examined with a warm stage, and the "mast-zellen" presently to be described need another stain to bring them out well. But with these exceptions all the points of any clinical significance are brought out by this very simple and easy staining method —one which can be done with sufficient accuracy in fifteen minutes, and which produces permanent specimens of the greatest value.

The following are the names usually given to the varieties of white cells present in normal blood, with the percentage of each variety in any 100 leucocytes:

1. Polymorphonuclear neutrophiles	60	to	70	per cent.
2. Lymphocytes	20	to	30	"
3. Large mononuclear and transitional cells	4	to	8	"
4. Eosinophiles	$\frac{1}{2}$	to	4	"
5. "Mastzellen"	$\frac{1}{10}$	to	$\frac{1}{4}$	"

This nomenclature is poor in many respects. It is based partly on doubtful theories of origin (as in "lymphocyte"), partly on appearances when stained in a certain way ("eosinophile," "neutrophile"), partly on theories of cell development ("transitional cells"), and partly on the number or shape of nuclei ("mononuclear," "polymorphonuclear"). It is cumbersome and does not explain itself. But, as no better has yet become current, we must for the present make use of it (Plate VIII.).

1. *Polymorphonuclear Neutrophiles.*—These are by far the commonest among the leucocytes, and form almost the whole of the cellular elements in ordinary pus;[1] they have twisted or broken nuclei, and have often gone under the name of "polynuclear leucocytes." As the nucleus is often single, the term is incorrect, and the more cumbrous word "polymorphonuclear" corresponds to the fact that the nucleus takes a very large number of shapes in different cells. No two are ever alike. When stained with Ehrlich's tri-color mixtures the protoplasm is seen to be crammed with very fine irregular granules.

The stain is not neutral, but acid, and these granules are not properly neutrophilic, but faintly acidophilic. Ehrlich's mixture is a differential acid stain with a rather slight affinity for this set of granules. They take a violet or pinkish color. The cell is actively amœboid, and has almost a monopoly of the function of emigration. As seen in the stained specimens, the cells are usually spherical, occasionally showing a pseudopod, and vary a good deal in size—averaging about 13.75μ

[1] The pus in the pustules and bullæ of pemphigus and some other skin diseases is composed wholly or largely of eosinophiles, and Senator reported the pus in the urinary sediment of a case of nephritis to be composed wholly of lymphocytes.

DESCRIPTION FOR PLATE VIII.

A. VARIETIES OF LEUCOCYTES AS SEEN IN NORMAL BLOOD.

1. *Small lymphocytes* (or lymph corpuscles).

   Note absence of granules. Nucleus may be light or dark. Protoplasm hardly visible. Cells about 5–10μ diameter; usually spherical. These cells are the youngest seen in the blood.

2. *Large lymphocytes.*

   No granules. Nucleus and protoplasm both stain feebly. Relatively more protoplasm than in small forms. Every size intermediate between No. 1 and No. 2 is to be found.

   The outline of the whole cell is more irregular than in No. 1.

3. *Transitional forms* (Ehrlich).

   Differ from last variety in that the nucleus is more or less indented; rarely a few purplish granules in protoplasm.

4. *Polymorphonuclear neutrophiles.*

   Note that granules vary in size and shape and are present over as well as around the nucleus. Average diameter of whole cell = 13.7μ. Nucleus always twisted and irregular.

5. *Eosinophiles.*

   Usually polymorphonuclear. Note loose arrangement of granules, their uniform and relatively large size, spherical shape, and copper color.

B. LYMPHATIC LEUCÆMIA.

1. *Myelocyte.*

2. *Polymorphonuclear neutrophile.*

   All the other white corpuscles present are lymphocytes and show the variations which this cell may present.

   Blood examination in this case showed—

Red cells	4,100,000 per c.mm.
White cells	92,000 " "

   Differential count of 1000 leucocytes showed—

Small lymphocytes	92 per cent.
Large lymphocytes	4 "
Polymorphonuclear neutrophiles	3 "
Eosinphiles	0.9 "
Myelocytes	0.1 "

   While counting these, 1 normoblast was seen.

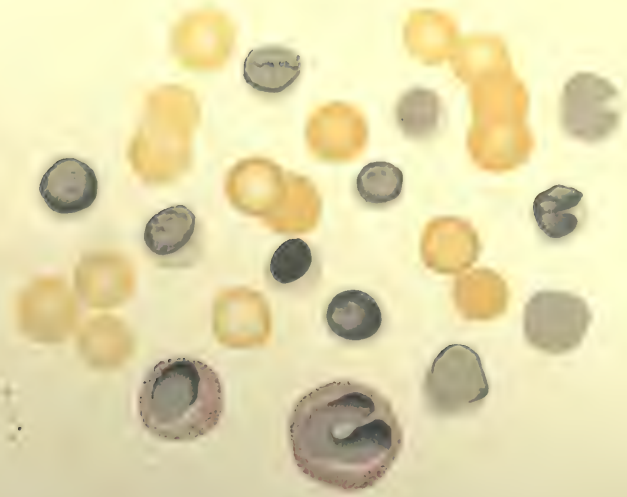
PLATE VIII.

FIG. A.

SCALE OF μ

0 10 20

FIG. B.



in diameter. Immersed in the pinkish granules above described, one or more neuclei are seen, which usually take in part a dark blue or greenish blue stain and in part a lighter blue. Owing to the twisting and diving of the nucleus in the cell body, the layer of granular protoplasm through which it is seen is much deeper at some points than at others. Sometimes it is so deep that the nucleus disappears altogether beneath the granules, reappearing again at a shallow spot farther on. This gives the appearance of one or more breaks in the nucleus in some specimens, while in others the fainter staining or shallower coating of granules over the nucleus lets the whole of it appear. The nucleus is often so bent on itself as to resemble the letters V, E, Z, C, or S. Or it may be in the form of a short, thick rod, straight or slightly curved or indented on one side. It seems to be more deeply buried within the cell body than that of the other neutrophilic elements in the blood (myelocytes), whose nucleus usually approaches very near the surface at some one or many points. As above mentioned, the staining of the nucleus is apt to be darker at some points than at others. Overheating or other mistakes in technique make these cells smaller. In examining stained specimens they are usually the prominent feature of the slide, both owing to their number (60 to 75 per cent. of all leucocytes) and the showiness of their pink and blue stain.

(2) Next in frequency of occurrence is the so called "*lymphocyte*" or "small mononuclear leucocyte," which is found in normal blood to the extent of 20–30 per cent. of all the leucocytes. Like all leucocytes, it is a spherical cell, and is usually described as varying in size from a little smaller to a little larger than a red cell (5–10μ), and having a deeply blue-stained nucleus which is covered by only a thin coating of protoplasm, the latter staining faint pink or else not staining at all, but appearing as a transparent ring lighter than the background. There are numerous variations from this type;[1] and, in fact, it is as common to see the nucleus pale blue as dark blue.

(3) In the "*large mononuclear*" cells above referred to as making up with the next variety about 4 to 8 per cent. of the white cells in normal blood, both nucleus and protoplasm stain very faintly, so that in a hasty examination their number may be underestimated. The pale blue nucleus is round or oval, and takes up but a comparatively small portion of the cell. The surrounding protoplasm stains light pink or not at all. The diameter of the cell is usually given as two to three times that of

[1] (*a*) *Variations in Size of Lymphocytes.*—There occur cells of every diameter between that of red cells and that of the "large mononuclear" varieties, and no method has yet been suggested by which a boundary could be set up parting the "large" mononuclear from the "small" mononuclear. It remains a matter of personal equation whether certain of these cells are classed under the one heading or the other. The colors do not help us, for the

(*b*) *Variations in staining properties* make it impossible to classify accurately by the stain. The nucleus may stain pale blue, like that of the "large mononuclear," or the protoplasm may not appear at all, not even as a vein of transparent substance; in which case it is very difficult at times to distinguish the cells from the extruded nuclei of nucleated red cells. The nucleus may stain dark in some parts and light in others, or show a clearly marked network of dark lines.

(*c*) The *nucleus* may be indented deeply or even divided in two, in which latter case it may be impossible to distinguish the cell from the polymorphonuclear cells whose granules do not stain, such as occur in some specimens, especially in leucæmia. The deeper layer of protoplasm round the nucleus in these latter is perhaps the best differentiating mark.

a red cell. The difficulty of separating them from the small mononuclear has already been noted. Thayer[1] has proposed to make the size of the nucleus the deciding point.

(4) "*Transitional cells*" are precisely like the last described, except in having a more or less deeply indented nucleus. Their percentage varies a great deal, but in normal blood is seldom over 3 to 4 per cent. They are so nearly like the last named variety that it seems best, in the absence of any known difference in the significance of the two, to class them together.

(5) "*Eosinophiles*" are usually the easiest of all leucocytes to be distinguished, owing to their showy, brilliant stain. They are about the size of a polymorphonuclear cell, and are either mononuclear or polynuclear. They are characterized by the presence of large spherical granules, which refract the light strongly and have a great affinity for acid stains like eosine. These granules average 1μ in diameter—much larger than the granules of the polymorphonuclear cells, from which they also differ in being spherical, while the latter are irregular in shape and size. With Ehrlich's stain they do not take the pure eosine pink, but rather a reddish brown or a port-wine color. Their percentage in normal blood is so small that we may hunt some time without seeing one.

(6) "*Mastzellen.*"—Very rare in normal blood, and best stained with a solution of dahlia, which brings out the basophilic granules. With Ehrlich's stain these cells may sometimes be recognized by the total absence of color in the granules, which appear like small vacuoles in the cell. "Mastzellen" have no known significance in the blood.

We have now described the appearance of the red and white cells of normal blood when stained by Ehrlich's method. If the slides are prepared by the same observer and with the same technique in all cases, a general idea can be formed from the number of red cells in a field of (say) a $\frac{1}{12}$ immersion lens with a No. 2 eyepiece, whether the whole number of red corpuscles is greater or less than the normal. Such a judgment rests on the fact that any one observer can learn to spread the layer of blood about the same thickness in each case.

In cases where the hæmoglobin is markedly dimished the pallor of the centre of the corpuscles attracts our attention. Variations in size, shape, and color of the red cells are easily seen. The discussion of these will be found later on (see p. 665), as also the significance and appearance of the various kinds of nucleated red cells. In estimating the percentages of the different varieties of leucocytes by the so called differential count, a mechanical stage is a great help and enables us to go over the whole slide without danger of counting the same corpuscles twice.

Starting in (say) the upper left hand corner of the cover-glass, turn the screw so as to pass the lens across to the upper right hand corner, counting all the white cells seen on the way and noting down the number of each variety. Then return from right to left a little farther down[2] toward the bottom of the cover-glass, following a course like that shown in Fig. 41, p. 644, and so on until the whole slide is covered or until 1000 leucocytes have been counted. We can be sure that we are

[1] *Johns Hopkins Report on Typhoid Fever*, 1893.

[2] "Down" means, here, toward the observer. and "upward" away from the observer.

not counting the same cells twice if, as we turn at the end of each "furrow," we move the slide so far "upward" that none of the corpuscles seen in the last field are visible.

Percentages based upon counts of five hundred cells are fairly accurate, but it is best, as a rule, to count at least one thousand cells from the best portions of the slide, where the different varieties are well stained and easily distinguishable from each other.

In estimating the number of nucleated red cells or of polychromatophilic forms (see p. 674) a good way is to note how many we see while counting a given number of white cells—say 1000. "While counting 1000 leucocytes" (*i. e.* in a certain area) "*x* megaloblasts, *y* monoblasts, and *z* polychromatophile cells were seen," is a good formula for general use, inasmuch as we cannot easily find, as we should like to do, how many red cells have passed the eye in the same area and how many *out of this number* are nucleated.

Bacteriological Examination of the Blood.—Sittmann[1] considers that results of value for diagnosis and prognosis are to be obtained by putting a syringe directly into a vein under strictest asepsis, and withdrawing at least 1 c. c. of blood, from which cultures are made. He got 23 positive results in 53 cases, finding

Pyogenic	Staphylococci	11	times;
	Streptococci	4	"
	Pneumococci	5	" (all in pneumonia);
	Mixed	3	"

The number of colonies growing from 1 c. c. of blood and the kind of organism present Sittmann considers valuable in prognosis of septic cases.

In gonorrhœal arthritis the gonococcus has been found and cultivated from the blood.

Classification of the Diseases of the Blood.

All our knowledge of the so called blood diseases is of the most fragmentary and unsatisfactory nature, and all our statements concerning them must therefore be considered as in the highest degree provisional and open to revision as soon as new light appears. The origin of the blood and the method of its reproduction and renewal are matters mostly of speculation.[2, 3] We are ignorant whether there are any diseases of the blood itself except the parasitic diseases like malaria, and whether morbid blood changes are causes or results of other organic lesions; we may doubt whether the changes both in the blood and in the (supposed) blood-making organs are not both of them due to some third factor, itself unknown.

Finally, the identity and individuality of the several "blood diseases" about to be described is open to considerable doubt.[4] In a few years we may have "changed all that." Pernicious anæmia and leu-

[1] *Deut. Arch. f. klin. Med.*, vol. liii., 1894.

[2] See (*a*) Neusser, *Wien. klin. Woch.*, 1892, Nos. 3 and 4; (*b*) Hayem, *loc. cit.*, pp. 589–610; (*c*) Weiss, *Wien. med. Presse*, 1891, p. 1537.

[3] Osler in *The American Text-book of Med.*, vol. ii. p. 185.

[4] Herz, "Blutkrankheiten," *Virchow's Archiv*, vol. cxxxiii.

cæmia may be found to be only different types of a single disease. Chlorosis may be classed with the other secondary anæmias, and Hodgkin's disease may be grouped with the malignant tumors or infectious diseases.

Still, we must keep to some nomenclature while a better is being evolved, and the following division of the morbid phenomena to be considered must serve us for the present:

1. Plethora.
2. Anæmia { (*a*) Primary anæmia { 1. Chlorosis. 2. Pernicious anæmia. (*b*) Secondary anæmia.
3. Leucæmia { Splenic-myelogenous. Lymphatic.
4. Leucocytosis.
5. Hodgkin's disease.
6. The blood in infancy.

PLETHORA AND POLYCYTHÆMIA.

The so called "plethoric habit" is usually an affair of vaso-motor origin or of venous stasis, and represents no real change in the constitution of the blood. Oertel, however, maintains that in obesity and uncompensated heart disease we may have a true overfilling of the vessels. There are certainly a few conditions in which the number of corpuscles per cubic millimetre *seems* to be increased when the element of stasis is excluded. These are—

1. In the newborn.[1]
2. In high altitudes.[2]
3. In delayed menstruation.[3]
4. In the cyanosis of congenital heart disease.[4]
5. In myxœdema.[5]
6. In phosphorus-poisoning.[6]
7. Whenever blood pressure is raised.

1. The plethora of the newborn will be mentioned later in the account of children's blood.

2. Persons who take up their residence in the high Swiss sanitaria soon show a marked increase in the number of red cells per cubic millimetre, the counts running often as high as eight or nine million cells per cubic millimetre, and the hæmoglobin rising proportionally. This condition has been carefully studied by Miescher, Wolf, and others, and the influence of stasis and concentration of the blood by such influences

[1] (*a*) *Zeit. f. Heilkunde*, 1890, vol. xii.; (*b*) Woino-Oransky, *Dissert.*, St. Petersburg, 1892. [2] Miescher, *Schweizer Correspondenzbl.*, Jahrgang xxiii. No. 24.

[3] Joslin and Denny, unpublished investigations at the Massachusetts General Hospital.

[4] (*a*) Gibson, *Lancet*, Jan. 5, 1895; (*b*) Carmichael, *Edin. Hosp. Rep.*, 1894, vol. ii.

[5] Kräpelin, *Deut. Arch. f. klin. Med.*, 1892, xlix.

[6] V. Jaksch, *Deut. med. Woch.*, 1893, xix.

as diarrhœa carefully excluded by experiments on animals. The following table illustrates these facts:

Location.	Observer.	Altitude in metres.	Number of corpuscles.
Christiana	Laache	Sea-level.	4,970,000
Göttingen	Schäfer	148	5,225,000
Tübingen	Reinert	314	5,322,000
Zürich	Stierlin	414	5,752,000
Auerbach	Wolff	425	5,748,000
Görbersdorf	Jaruntowski	561	5,800,000
Reiboldsgrün	Wolff	700	5,970,000
Arosa	Egger	1800	7,000,000
Cordilleras	Viault	4392	8,000,000

The explanation of this form of polycythæmia is still in dispute, and no satisfactory theory has yet been advanced.

On returning to the valleys the blood becomes normal again within a few months.

3. In nine successive cases where the menstrual appearance was delayed some months or more we have noted a decided increase in the number of red cells, the influence of stasis being excluded.

4. The cyanosis of congenital heart disease is not always accompanied by a true venous stasis, and the increase of red cells which has been noted by several observers and by ourselves is not easily explained. The same is true of the plethora of myxœdema and phosphorus-poisoning.

Exercise, massage, electricity, or any influence which temporarily raises blood pressure concentrates the blood by driving serum into the perivascular spaces, and so increases the count of cells per cubic millimetre.

Concentration of the blood by the diarrhœa of cholera or other severe diarrhœa is sometimes very great.[1] Vomiting, sweating, and the rapid accumulation of serous effusions have a similar effect.

Occasionally we meet with cases of simple cyanosis not to be explained by any of the ordinary causes and apparently of vaso-motor origin.[2] In a recent case of this kind we counted ten million of red cells in a cubic millimetre. Autopsy showed no explanation of the cyanosis.

We must bear in mind the effect of all these influences before making inferences from a single drop as to the condition of the whole blood.

ANÆMIA.

DEFINITION.—A deficiency in corpuscle substance—*i. e.* in red corpuscles—in hæmoglobin, or both. True anæmia, like true plethora, is far rarer than is popularly supposed. Probably a majority of the patients called anæmic on the evidence of the color of the skin are not anæmic at all. Nothing is more deceptive. We may have anæmia with a good color in the face, or (what is more common) we may

[1] Sadler, *Fortschritt. de Med.*, Supplementheft, 1892.
[2] Vaquez, *Bull. de Méd.*, Paris, 1892, No. 38.

examine the blood of persons who look almost bloodless and find a normal number of red cells and slight, if any, deficiency in hæmoglobin. The most striking example of this is in the so called tropical anæmia, lately studied by Crijns,[1] Eykmann,[2] and Glogner,[3] which affects almost all persons born in cooler countries who take up their residence in the tropics. The pallor, both of skin and mucous membranes, is often intense, but the blood is found to be normal in all respects. In this country the color of the lips and of the inside of the mouth is a far better guide than that of the skin, but even this occasionally misleads us, especially in tuberculosis and malignant disease.

The division of cases of anæmia into *primary* and *secondary* is, like most of our statements concerning the blood in disease, a rough and ready one and open to many objections, but it has yet a certain utility if not used to mean more than this—viz. that in some cases (the so called "*secondary anæmias*") the most important and influential etiological factor is apparently not a lesion of the blood or bloodmaking organs, but rather some disease like cancer or malaria; while in "primary anæmia" the important though not the only causal influence is unknown or resides in some hypothetical lesion of bloodmaking functions of the organism.

Perhaps the prolonged drain upon the system of a disease like syphilis or a chronic suppuration may eventually produce actual lesions in the bloodmaking organs which persist after the removal of their cause, so that the resulting anæmia might be called "primary." Still, the most important factor may have been the suppuration or the syphilis.

On the other hand, in cases of chlorosis or pernicious anæmia there may be in the history a nervous shock, some malaria or some hemorrhage—say from long-standing hemorrhoids—and yet the anæmia be properly called primary in case these factors are not of sufficient severity to produce any considerable blood trouble in a moderately sound individual. A few malarial chills or the bearing of a child is not sufficient, in itself, to produce any considerable anæmia, although if there be a very great natural lack of vigor in hæmogenetic function it may be that malaria or parturition will act as the straw that breaks the camel's back. Such cases are to be called primary, since the unknown factor seems to be the principal one, and the known factor, such as malaria, does not seem of itself adequate to produce such severe symptoms.

It is true that there is usually a certain amount of difference both in the nature and in the severity of the symptoms in the two classes of anæmias, and also, in the vast majority of cases, a different blood constitution. It may be that we have already in our possession criteria in the appearance of the blood itself to enable us to distinguish pernicious anæmia from any secondary anæmia, but certainly in regard to chlorosis the blood alone is not sufficient to give us the diagnosis.[4]

In such divisions, therefore, as we find it best to make between the different varieties of anæmia our appeal must be made partly to the etiology, the symptoms, and the course, as well as to the blood itself, though in many (perhaps most) cases it is the blood examination that

[1] *Virchow's Archiv*, vol. cxxxix. [2] *Ibid.*, vol. cxxvi. [3] *Ibid.*, cxxvi. and cxxxii.
[4] Cf. Birch-Hirschfeld, *Eleventh Cong. f. Innere Med.*, Wiesbaden, 1892.

puts us on the right track, and its indications may often override and reverse the judgments we should have made without it (see p. 665).

Bearing these facts in mind, we may classify the anæmias as follows:

1. Primary anæmia { (*a*) Chlorosis. (*b*) Pernicious anæmia.
2. Secondary anæmia (due to cancer, tuberculosis, bad hygiene, etc.).

Such terms as "simple primary anæmia" and "splenic anæmia" are misleading, and should be discarded. The cases classed as "simple primary" anæmia belong under the heading of anæmia due to bad hygiene, adolescence, or other cause—*i. e.* they are secondary anæmias—while the term "splenic anæmia" refers simply to the association of anæmia with splenic enlargement, such as occurs in malaria, rickets, syphilis, Hodgkin's disease, and other conditions, and represents no single clinical or pathological picture.

Primary Anæmia.—(*a*) Chlorosis.

The "greenish" tint in the pallor whence the name chlorosis is derived is not a very constant nor a very prominent feature of the disease. It is said to be more marked in the rare cases in which the disease occurs in brunettes.

The disease is to be recognized rather by its almost exclusive occurrence in young women between the ages of seventeen and twenty-three, by the presence of the symptoms of a considerable anæmia without loss of flesh, and by the relatively large diminution in hæmoglobin without any other striking changes in the blood.

Etiology and Pathology.—Similar blood changes occurring in other diseases are sometimes known as "*chloranæmia*." Few diseases have given rise to a greater speculative activity in the search for a theory of origin and nature, and in few have the results been less conclusive. Briefly stated, the principal theories in regard to it are as follows:

1. That it is due to a hypoplasia of heart and aorta (Virchow).[1]
2. That it is due to a hypoplasia of heart, aorta, and genital organs (Rokitansky).[2]
3. That it is due to a fæcal absorption and poisoning from constipation (Sir Andrew Clarke).[3]
4. That it is due to the formation of ptomaïnes in the intestine, which prevents the normal formation of hæmoglobin there (Forscheimer).[4]
5. That it is due to defective development of red cells (Hayem).[5]
6. That it is an infectious disease (Clements).[6]
7. That it is due to a neurosis (Murri).[7]
8. That it is due to gastroptosis from tight lacing (Meinert).[8]
9. That it is the result of the drain on the blood caused by the

[1] *Gesammte. Abhandl.*, 1856, p. 494. [2] *Handbuch d. Path. Anat.*, 1846.
[3] *Lancet*, 1887, ii. 1003. [4] *Assoc. of American Physicians*, April 24, 1893.
[5] *Loc. cit.*; also *Mercredi Méd.*, April 24, 1895.
[6] *Cong. de Méd. int.*, Lyons, Oct. 25, 1894.
[7] *Internat. Cong. d. Scien. méd.*, Rome, Mar. 29, 1894.
[8] *Versaml. d. Gesell. f. Kinderheilk.*, Nürnberg, 1893, p. 43.

establishment of the function of menstruation in women whose hæmogenetic power is naturally feeble (Lloyd Jones).[1]

The following factors have also to be considered in etiology:

(*a*) The important influence of *cold*, which has been well brought out by Murri.[2]

(*b*) Jolly[3] has tried to show that it is closely allied to the tubercular diathesis, and occurs chiefly in those who have a tubercular family history.

(*c*) There seems to be no doubt that bad hygiene, homesickness, and lack of adaptation to a new climate have a considerable importance.[4]

(*d*) Oppenheimer[5] believes that the presence of adenoid growths in the naso-pharynx is frequently responsible for the disease.

(*e*) Heredity probably plays a certain part in its causation. Certainly, we often see several members of one family attacked by it.

The fact that permanent cure is the rule militates against the idea that any anatomical changes in heart, aorta, or genitals account for the symptoms given; for these lesions are not to be removed by iron.

The absence of any increase of sulphates in the fæces is against the theories of Clarke and Forscheimer, and the evidence given by the latter of hæmoglobin formation in the intestine is altogether insufficient.

Hayem's theory is little more than a statement of the difficulty to be explained, except in so far as it asserts that the blood changes are primary, and of this the evidence is unsatisfactory, since all the same blood changes occur as secondary to malignant disease and other causes.

The assumption of its infectious nature, based on the presence of an enlarged spleen and a pyrexia in certain cases, cannot be categorically denied, but the symptoms on which it is based are frequently absent, and are not sufficient in the absence of other evidence to warrant the hypothesis.

A disturbance in the working of the vaso-motor system is certainly characteristic of the disease, and may afford some insight into its nature without very adequately providing us with a cause. The gastroptosis observed by Meinert in certain cases would not be removed by the ingestion of iron, and cures by the simple use of this metal would be inexplicable. Tight lacing is certainly absent in the history of many well marked cases.

The theory of Lloyd Jones is more plausible than most in that it attempts to account for the very important fact that the disease is practically confined to young women soon after the establishment of the menstrual function. The defective hæmogenetic power which must be assumed as the basis of the theory is little more than a name, considering our present uncertainty as to how and where the blood is made. Still, it seems to us that this theory answers tolerably well to state our present knowledge (or ignorance) about the antecedents of the group of symptoms we call chlorosis.

That heredity, cold, tuberculosis, mental strain or shock, bad hygiene, the "third tonsil," and change of climate each play a certain part in

[1] *Brit. Med. Soc.*, Aug., 1892.
[2] *Loc. cit.*
[3] *Thès. Doct.*, Paris, 1890.
[4] Osler, *Amer. Text-book of Med.*, vol. ii. p. 197.
[5] *Berl. klin. Woch.*, Oct. 3, 1892.

some cases we think there is evidence for believing, but there are many other cases in which they form no factor.

Symptoms.—The remarkable uniformity of type in this disease is noteworthy. A girl begins gradually to get weak, tired, dyspeptic, and pale; then notices shortness of breath on exertion and drowsiness in the daytime, although often the nights are poor. Headache is very frequent, and œdema of the ankles almost as much so. Palpitation is almost invariable, and ringing in the ears and dark spots before the eyes are very common. It is sometimes startling to hear one girl after another repeat these symptoms as if they had read them in a text-book of medicine. Anorexia is very constant, and nausea and vomiting not uncommon. Perverted appetite is not rare. Constipation, if present, is usually chronic, antedating the symptoms. It is *not* very frequent in our experience. The menses are usually absent or very scanty and painful, and pregnancy is not infrequently suspected, especially in those who suffer from vomiting. Pains in the back and groins are often complained of, and leucorrhœa is common. By far the larger number of our cases have been in domestic servants and working girls, and very few among the well-to-do classes. A considerable number of cases dated from a change of residence from Ireland or Canada to New England. There have been no cases coming on suddenly after a psychic disturbance such as have frequently been mentioned by French writers and others, a gradual onset being the rule in our experience. The ages have ranged very closely between sixteen and twenty-five,[1] and there have been no cases in males. Fever has occurred in most of our cases, ranging from 99° to 100° F. Anomalies of the genitals have not been noted in any case. The patients have not seemed to have been addicted to tight lacing, nor has tuberculosis been abnormally frequent in the family history. There are many symptoms pointing to a derangement of the vaso-motor and sympathetic nervous system. The frequent flushing of the face, the sweating and coldness of the extremities, the mottling of the skin, the abnormal sensitiveness to cold, the hypersecretion of the digestive fluids and of pale urine of low specific gravity, the capricious, sometimes depraved, appetite, and the neurotic or hysterical mental condition, form a group of symptoms to which the explanation applies, and the constipation is referred by some writers to the same cause.

Physical Signs.—As before mentioned, the color has seemed to us to be usually of various shades of white to whitish yellow, rather than green. The "sky blue" sclerotics described by Osler[2] have appeared rather as a pale china blue, perhaps like the sky very near the horizon. Patients often blush easily, and the color may stay for some hours. The color of the gums and palpebral conjunctivæ may be almost gone. The tongue is usually coated and the breath foul. The heart is occasionally enlarged, but in a number of our cases the apex was found in the fourth interspace inside the nipple, suggesting the hypoplastic heart of Virchow. Over the whole cardiac area a soft systolic murmur is almost invariably to be heard, loudest at the pulmonary orifice. Some-

[1] Rieder (*Münch. med. Woch.*, 1893, p. 225) reported 3 cases of typical chlorosis, beginning at thirty, forty, and forty-two years of age, respectively.

[2] *Loc. cit.*

times the murmur can be heard in the left axilla and back. In rare cases the murmur is harsh or musical. The pulmonic second sound is accentuated. Over the jugulars, particularly the right, a steady roar or hum is generally audible (*bruit de diable*). The heart's action, though accelerated by slight exertion or excitement, is usually regular. The lungs show nothing abnormal.

Dilatation and downward displacement of the stomach appears to be not uncommon.[1] The spleen is occasionally somewhat enlarged. Œdema of the ankles is the rule. The uterus is frequently retroverted. The patients are usually well nourished. The hands and feet are usually cold and clammy, and mottling of the skin or even a "tache cérèbrale" is often to be seen. Areas of pigmentation about the joints are mentioned by some writers, but we have never observed them. Moderate enlargement of the thyroid gland is stated by Stengel to be very common. Slight exophthalmia may occur.

Examination of the stomach's contents in the dyspeptic cases shows usually an increase, and very rarely any decrease, in the amount of HCl.[2]

The urine is usually pale and of low specific gravity, otherwise not abnormal. The fæces show no increase in the products of decomposition,[3] and are not specially foul smelling.

The temperature is usually normal or slightly elevated. The nervous system shows none of the changes so common in pernicious anæmia. The knee-jerks are normal or increased, and anæsthesiæ and paræsthesiæ are rare.

Thromboses are not infrequent,[4] and death has repeatedly been reported from plugging of a cerebral sinus. The femoral veins are a more frequent seat of thrombosis. This tendency is in marked contrast with the condition obtaining in pernicious anæmia, where the blood is abnormally slow in clotting.

The Blood.—The pallor and fluidity of the drop as it emerges from the point of puncture is sometimes as marked as in pernicious anæmia. The technique must be very quick if we are to avoid clotting.

The red cells are usually very little diminished in number, rarely falling below 3,500,000. Occasionally they may sink below 2,000,000, but this is very exceptional. As in secondary anæmias, the hæmoglobin suffers chiefly, being diminished usually to less than one half the normal amount. In 77 cases observed by us the corpuscles averaged 4,050,000 per c. norm. and the hæmoglobin 41 per cent. Thayer's 63 cases averaged 4,096,544 corpuscles, with 42.3 per cent. hæmoglobin.

V. Jaksch[5] has shown that chemically it is the albuminous substances that suffer, their deficiency running parallel to the want of hæmoglobin.

The white cells are usually normal in number. Occasionally there is slight leucocytosis for a time. The differential count shows often an increased percentage of lymphocytes, even as high as 40 per cent., the polymorphonuclear varieties suffering proportionally. The other varie-

[1] Meinert, *loc. cit.*

[2] Cantu (*Dissert.*, Pavia, 1893) verified this in 29 cases; Gallot, *Paris Thèsis*, 1894; Oswald, *Münch. med. Woch.*, 1894, No 27.

[3] Raethers, *Dissert.*, Berlin, 1891.

[4] Kochel, *Neurolog. Centralbl.*, Aug., 1894.

[5] *Cong. innere Med.*, 1893, p. 239.

ties are not specially affected. Small percentages of myelocytes have been reported by Hammerschlag and by Neusser[1] in severe cases.

The blood plates are said to be increased in number. Deformities in the shape of the corpuscles are very common, and in extreme cases may be as great as are to be found in any condition. The average diameter of the corpuscles is usually diminished—sometimes very much so, contrasting markedly with the increase in the diameter characteristic of pernicious anæmia.

The pallor of the centres of the stained cells is proportional to the lack of hæmoglobin. Nucleated red corpuscles are sometimes present in small numbers. They are of the normoblast type.

COMPLICATIONS.—Gastric ulcer, phthisis, Graves' disease occasionally accompany or follow chlorosis.

PATHOLOGICAL ANATOMY.—So few cases die of the disease that we know hardly anything about its pathology. Hypoplasia of the heart with a thin, relatively narrow aorta occurs in some cases. In others the heart is dilated and the left ventricle hypertrophied. Anomalies of the genitals have been mentioned.

DIAGNOSIS.—We need to exclude by means of etiology and physical examination the anæmia secondary to tuberculosis, nephritis, and bad hygiene, in which the blood condition may be similar, and pernicious anæmia, which may be identical with chlorosis in everything except the condition of the blood.

Pernicious anæmia has usually a much greater reduction of red cells, a higher color index, a greater average diameter to the red cells, and, above all, a majority of megaloblasts among the nucleated red corpuscles present.

Doubtless many cases are called chlorosis on account of simple pallor where a blood examination would show no lack of red cells or hæmoglobin.

PROGNOSIS AND COURSE.—The majority of cases yield readily to treatment. It appears, however, that some cases in the New England States are more obstinate than those seen in Baltimore. It cannot be said of our cases that iron is a real specific in each and every patient. Occasionally we have seen patients fail to show any improvement after several months' treatment under the most favorable conditions. Relapses are not uncommon, especially if the treatment be not continued for considerable periods. Death may result from thrombosis[2] of a cerebral sinus or very rarely from the anæmia itself. Transitions to pernicious anæmia are probably legendary.

TREATMENT.—All severe cases should be confined strictly to bed. It has been shown by Murri[3] that exposure to cold in any form aggravates all the symptoms notably, a cold bath being followed by a marked fall in the number of red corpuscles and increase of urinary pigments, sometimes by true hæmoglobinuria.[4] Most cases are worse in winter. This indication must be borne in mind, and if the weather be at all cold even convalescents should be kept in the house and all cold bathing avoided. Such patients do not need exercise,[5] and are very easily

[1] *Wien. klin. Woch.*, 1892, p. 42.
[2] Osler, *loc cit.*, vol. ii. p. 197.
[3] *Loc. cit.*
[4] Noorden, *Berl. klin. Woch.*, Aug. 20, 1894.
[5] Hayem (*Revue d. Méd.*, April 24, 1895) advises *no exercise whatever* in chlorosis.

tired by it. The maximum of nutrition should be aimed at, and the bowels kept regular, if necessary with a laxative.

After giving a thorough trial of the method of treating the disease simply with a laxative and also to the intestinal antiseptics recommended by Forscheimer, we believe it is really susceptible of proof that these drugs cannot compare in efficiency with iron given in the form of Blaud's pill. In a large number of cases we have watched the hæmoglobin percentages remain stationary for weeks under laxatives, intestinal antiseptics, or both, and then rise quickly to normal under Blaud's pill, gr. xv, t. i. d. At a recent discussion among the greatest German authorities on anæmic conditions there was surprising unanimity of belief that Blaud's pill far exceeds all other preparations, old and new, in the treatment of chlorosis.

Not every case responds to it, but the vast majority do, and it is not at all constipating. Sometimes the addition of arsenic in the form of Fowler's solution is of service. A point of interest observed by us in a few cases was the marked improvement under Blaud's pill of patients who looked anæmic, but who were not so according to the blood count of hæmoglobin estimate. It has been observed by Townsend[1] that cases treated by Blaud's pill after a course of intestinal antiseptics were more benefited by the iron than those where no antiseptics were used, although the latter alone had hardly any effect on raising the hæmoglobin percentages.

Bleeding[2] as a means of stirring the inert bloodmaking functions has been successfully practised by Wilhemj, Scholtz, and Rubinstein, Scholtz having tried it in 200 cases. V. Noorden and other writers have been less uniformly successful, and consider that it requires great skill to apply the treatment to the right cases and at the right period of their course. If we hit just the right moment, it may be of great service; otherwise it is harmful and dangerous. We have no personal experience with this method of treatment.

Lavage of the stomach[3] has been practised by Pick under the idea that the disease is due to the absorption of products of gastric fermentation. He claims great success for this method.

The correction of obvious errors in hygiene is of course essential, and some cases need no further treatment.

Sodium bicarbonate, combined with gentian or nux vomica, is very efficient for the indigestion in many cases.

For the constipation nothing is better than the extract of cascara sagrada in pill form, 1 to 3 grains daily. Many patients need no laxative while taking Blaud's pills, which, so far from being constipating, seem to have a slight laxative tendency.

Change of surroundings or climate is very helpful in many cases.

(*b*) Pernicious Anæmia.

Definition.—A profound and almost invariably fatal anæmia without adequate known cause, characterized by an extreme diminution in

[1] *Boston Med. and Surg. Journal*, 1896.
[2] Wilhemj, *Blutsucht und Aderlass*, Guestrow, 1890.
[3] Pick, *Wien. klin. Woch.*, 1891, p. 939.

the number of red corpuscles and other changes in the blood, as well as by the presence of fatty degeneration of various organs, and usually by an absence of emaciation and a tendency to spontaneous but temporary improvement, followed by relapse.

(*a*) Fatality is not in itself the distinguishing mark of the disease. Chlorosis sometimes kills, and certain cases identical clinically with pernicious anæmia have recovered after expulsion of intestinal parasites. Yet in this country and with our present ignorance of its cause it is a fatal disease. Except the cases just mentioned, where recovery has followed the expulsion of parasites, the reported cures are almost invariably doubtful, either from lack of a careful blood examination, such as would put the diagnosis beyond doubt, or of a sufficient period of good health to justify the term "cure." The cases are reported when just on the top of a wave of improvement, and the subsequent relapse is not followed. Of the 60 cases known to us, not one is now known to have lived over three years; 52 are known to have died within two years of the time when they first came under observation; the others have been lost track of.

(*b*) Absence of known cause is not sufficient to justify the diagnosis, for we are equally in the dark as to the etiology of chlorosis and of other milder forms of anæmia. It is, none the less, a constant mark of the disease, which it shares only with chlorosis, that in the etiology the important factor is the unknown factor.

(*c*) It is very possible that we have in the characteristics of the blood itself a sufficient criterion for making the diagnosis without any other knowledge.

There do, indeed, seem to be certain features in which the blood constitution is unique, but these characteristics are so minute and rest on so small a number of cases observed since Ehrlich's methods were introduced that we may rightly suspend judgment until a larger body of accurate statistics on this point has accumulated.

Pernicious anæmia is not a very uncommon disease: 47 cases have come under our personal observation within the last three years, and doubtless a more careful examination of the blood as a matter of routine would result in the diagnosis being made more frequently than is now the case. These cases, together with 19 others occurring in the Massachusetts General Hospital within the last ten years, give us a group of 66 cases which are used as a basis for most of the conclusions of this article. The experience of others has not, however, been disregarded, although names have, as a rule, been omitted.

ETIOLOGY.—*Age.*—The disease seems to be more incident to middle life. Of 66 cases known to us, 46 occurred between the ages of thirty and sixty, the remainder being about equally distributed above and below those ages. It is not uncommon in infancy, though as occurring at that age its type differs considerably from that seen in adults (see "The Blood in Infancy," p. 700). In 1 of our cases the symptoms began at the age of seventy-nine.

Sex.—Men are more frequently attacked than women: 45 of our cases were males and 21 females. Larger statistics show less difference than this between the sexes, but usually a preponderance of males.

Season, nationality, residence, and *occupation* appear to be of no spe-

cial significance in the disease, except in so far as residence in a place where the below mentioned parasites are found may be considered to favor its occurrence. A large number of cases has recently been reported in Ceylon among coolies, many of whom, to escape work, endeavored to make themselves sick by eating dirt. A large proportion of our cases were in well-to-do people.

In 3 of our 21 female cases the symptoms dated exactly from the time of the *menopause*, and in 1 case the complaints began nine weeks after *parturition*. In the others there was no known connection with activity or inactivity of the sexual organs, nor with any physical, mental, or emotional strain. There seems to be no good reason for supposing that the menopause or the parturition in these cases had any other etiological significance than as rendering the system somewhat less resistant to the unknown morbid influence to which the disease was due. There is even less reason to suppose any connection between the mild attack of *malaria* which occurred in 3 other cases (respectively eight, ten, and twenty years before the anæmia). *Syphilis* was not a factor in any case, so far as could be ascertained. *Intestinal parasites* (the bothriocephalus latus and the anchylostoma duodenale) have never been observed in this country in connection with the disease,[1] and our experience is no exception to this statement. In Germany and Egypt the group of cases associated with these parasites forms the only exception to the rule that a diagnosis of pernicious anæmia means death usually within two years, and always within four years. The expulsion of the parasites has been followed by genuine cure in some cases carefully studied by Schaumann and Askanazy.

Atrophy of the gastric tubules has not been observed in any of the cases collected by us. It has been mentioned by Flint, Fenwick, and others as occurring in association with the disease—whether as cause or result is doubtful.

Hemorrhages small in amount and extending over considerable periods were associated with 5 of our cases before the other symptoms of anæmia were marked. They may have been an early symptom of the disease itself. They were never more profuse than occur (*e. g.* from piles) in many persons over much longer periods without being followed by any considerable anæmia. The surgical records of any hospital furnish plenty of such examples. Cases are also mentioned where the symptoms of a pernicious anæmia dated immediately from a single large loss of blood. No such case is known to us.

So far as our experience goes, therefore, the etiological factors of this disease are entirely unknown. Even the apparently direct connection between the intestinal parasites and the disease is open to some doubt. For not every person who carries these organisms about with him gets pernicious anæmia or even any anæmia at all.

Pathological Anatomy.[2]—The preservation of the subcutaneous fat tissue is a very constant feature. It was present in 59 of our cases. Two cases verified by autopsy were decidedly thin, but no thinner than they always had been. The bright red color to the muscle fibre men-

[1] Except in very recent immigrants (see Vol. III., Intestinal Parasites).

[2] All the changes here recorded are also to be found in secondary anæmia, and have been produced experimentally by subjecting animals to repeated hemorrhages.

tioned by most writers was noted in all of our autopsies (unfortunately, only 10 in all).

Fatty degeneration and great pallor of all the organs is the most striking feature, and is most marked in the heart, the papillary muscles especially being spotted with small yellow areas, which have caused the somewhat far-fetched comparison to a tiger lily. The heart is not enlarged, but the flabbiness of its walls is notable. The blood remains fluid for a comparatively long period after death, and cover-glasses prepared from it show no considerable changes from the ante-mortem appearances. Serous effusions and local œdema may be found.

Punctate hemorrhages are more common in the serous surfaces than subcutaneously, but in a majority of our autopsies were very scanty. The surface of the stomach is in some cases remarkably smooth and shining, corresponding to the atrophy of the glandular structure. This was not observed in any of our cases. Spleen and liver are occasionally enlarged, especially in infants. No microscopic changes of importance in the spleen have been described. The liver is usually fatty, and shows in many cases the excess of iron on which so much stress has been laid by Hunter. This was present in 3 of the 5 autopsies of our series in which it was specially looked for. The kidneys are often fatty and contain an excess of iron.

The glands and the marrow of the long bones may show no change. They may present a deep red "currant jelly" appearance throughout, or there may be simply the "lymphoid" marrow common to this and many other conditions. The spinal cord not infrequently shows areas of sclerosis, particularly of the posterior columns, but in some cases of the lateral or anterior columns as well. The cervical enlargement shows most change. The gray matter is not affected.

Symptoms.—The gradual and insidious onset is notable. Patients can rarely fix any date for the beginning of their symptoms. Friends notice they are looking pale or "jaundiced" often before there are any symptoms at all. Muscular weakness and dyspnœa are the earliest symptoms, as a rule, and generally remain the most prominent ones throughout, sooner or later confining the patient to bed. Gastro-intestinal disturbances are next in frequency. Anorexia is a very early and constant symptom, though one eighth of our cases had a good appetite most of the time, and one man said he "never could get enough to eat." Periods of nausea and vomiting, occurring often without special relation to the time or character of the food, were present in five sixths of our cases, and at times were the most prominent feature of the clinical picture. But the fact that in almost all cases there occur periods of reasonably good digestion militates against the presence of any organic changes in the stomach, such as atrophy of its secreting tubules.

The bowels were loose in one third and constipated in one third of our cases, regular in the remaining one third. Diarrhœa is a very prominent feature in some cases, and is remarkably little affected by any treatment save opiates. Occasionally the discharges contain a little blood, but this is not particularly common.

Circulatory symptoms are, as a rule, confined to increasing dyspnœa on any exertion (which does not, however, get to the point of orthopnœa

and does not persist when the patient is at rest) and to œdema of the feet and legs. Such œdema occurred in two thirds of the cases observed by us, generally as a late symptom and not a prominent one. Occasionally the face is swollen also. In the 4 cases of our series in which this symptom was present, it first appeared late in the course of the disease. Ascites was noted in 2 and hydro-thorax in 3.

Palpitation on any exertion is as common in this as in other severe anæmias, and the same may be said of vertigo and faintness. These latter symptoms were the earliest noticed by the patient in 2 of our cases.

Headache is usually the only form of pain occurring in the disease. It was present in one fourth of our cases, and was severe and persistent in 2 of them.

The absence of any other pain is a notable point in the disease, though 2 of our cases had severe neuralgic pains in the arms during exertion, and only then.

Chills with sharp rise of temperature to 103° F. or more, and a fall to normal within twenty-four hours, occurred occasionally in 5 of our cases. No cause could be assigned for them.

Hemorrhages occurred in one third of our cases while under observation. Epistaxis was the commonest form, and bleeding gums the next in frequency. Subcutaneous hemorrhages (purpura) were noted in only 3 cases of the 66; 2 cases showed severe purpura hæmorrhagica. Retinal hemorrhages were present in 9 of the 11 cases in which they were looked for, and it is interesting to note that in none of these were there any hemorrhages elsewhere; 1 case showed marked hæmaturia for a few days.

Symptoms referable to the Nervous System.[1]—Attention has been called of late years to the not infrequent occurrence of symptoms pointing to the spinal cord or peripheral nerves in connection with pernicious anæmia. Such were present in more than one third of our cases. Numbness, tingling in the extremities, and pain along nerve trunks occurred in 9 cases, and symptoms of posterior spinal sclerosis in 5 cases, 1 of which showed well marked lancinating pains. A spastic gait with increased reflexes was noted in 1 case. There were also 2 cases (both females) in which symptoms of diffuse myelitis, with complete paralysis of the extremities and relaxed sphincters, was present in the last few months before death. Spasms like those of tetany were observed in 1 case. Aphasic seizures of short duration occurred in 2 cases, preceded in 1 case by complete coma lasting two hours. Hemiparesis, including the face and accompanied by inability to swallow, occurred in 1 case, gradually passing away within three days. Atrophy, weakness, and numbness of the left arm and leg, with tremor of the left hand, were features of 1 case. Mental symptoms are usually confined to a sluggishness gradually deepening into coma, in which the last two or three days of life are passed. But delirium, especially at night, is not very uncommon, occurring in 4 of our cases in the last week of life. In 2 cases there were well marked delusions and hallucinations of sight and hearing for two months before death. Symptoms of organic nervous disease were twice as common in women as in men in our series of cases.

Physical Examination.—All writers speak of the notably well

[1] Burr, *University Med. Mag.*, Phila., April, 1895.

preserved layer of subcutaneous fat, and our experience is entirely in accord with the observation. Emaciation occurred in only 7 of our cases, most of them individuals who had always been spare.

The color and appearance of the skin and mucous membranes are of importance. Greater pallor of the skin is not infrequently observed in chronic nephritis and other conditions. It is the quality of the pallor and the color of the mucous membranes that are important in pernicious anæmia. Nowhere do we see such whiteness of the palpebral conjunctivæ, of the gums, and nails. The skin is usually called lemon yellow, and this is the term set down in the records of almost all our cases. In reality it is a much lighter color than that of a lemon and nearer that of a grape-fruit. But the striking thing about it is the evenness of the color throughout the smooth skin, the color being the same in all parts of the face, as a rule, without variation on the cheeks or forehead.

The conjunctivæ are usually quite clear, but occasionally have a slight subicteric tinge that makes us look to the stools and urine to exclude an obstructive jaundice. The smooth, even color is different from the somewhat uneven or muddy pallor of anæmia secondary to malignant disease or cirrhotic liver.

The tongue has been noted to be unusually smooth and shiny in 5 of our cases; as a rule, it is clean. The gums bled easily in one third of the cases; often the patient could, by sucking, draw blood out of them. Occasionally the tissues of the mouth get into a very unhealthy condition with ulceration, oozing, bleeding surfaces, and looseness of the teeth as in scurvy.

The heart is rarely enlarged. The systolic murmurs commonly heard in all forms of severe anæmia, especially at the base of the heart, are perhaps louder and sometimes harsher in pernicious anæmia than in other forms. Occasionally to-and-fro murmurs are to be heard, although the autopsy shows no valvular lesion, and in 1 case there was from time to time a well marked presystolic murmur at the apex, which, however, was sometimes absent for weeks at a time. As a rule, the murmurs are not transmitted to the axilla or back, but there are a few exceptions to this.

The pulse has been not infrequently observed to resemble the Corrigan or "water-hammer" type. This was noted in 6 of our cases, in 2 of which there was also a "pistol-shot" sound in the peripheral arteries, the heart itself showing no sign of aortic regurgitation. Capillary pulsation is occasionally to be seen in the mucous membrane of the lips.

The lungs occasionally show the moist râles of passive congestion, and hydro-thorax may occur.

The liver is not infrequently somewhat enlarged, extending in 4 of our cases two fingers-breadths below the ribs and as high as the fourth rib, and in 2 others being considerably enlarged.

The spleen is usually not enlarged, yet it was palpable in 3 of our cases, once extending three and a half inches below the ribs.

Tenderness of the long bones is probably not more frequent in this disease than in various other conditions; it was noted in only one eighth of our observations.

Fever is the rule, and was present in all our cases, generally between 99° and 101° F.—a continuous fever—but occasionally ranging higher.

The urine is usually pale and of low gravity, despite the contrary observations of Hunter in certain cases. In only 3 of our 66 cases was it high colored at any time, and never so continuously.[1] The gravity averages 1015. Albumin and casts were present only in 4 cases, all over fifty, and amounted to nothing more than is common at that age in healthy persons. Peptonuria is frequently observed.[2] Temporary hæmaturia occurred in 1 of our cases.

The Blood.—Naturally our interest is centred here. The difficulty experienced in getting any blood at all from a puncture of the finger in these cases makes one wonder how the tissues are nourished as well as they are. From the ear it flows with little difficulty. To the naked eye its pallor is not more marked than in some cases of chlorosis and other less severe anæmias. Not infrequently we see a streaky or parti-colored look of the drop as it emerges. It is very slow of coagulation and runs quickly off the ear like alcohol.

Cases as they come under observation usually show a little more or a little less than 1,000,000 red corpuscles per cubic millimetre, but this sooner or later may fall to or below 500,000. It is astonishing how well some patients are for weeks at a time with only one tenth the normal number of corpuscles, while other patients with double the number of cells will be much sicker. Evidently it is not simply the quantitative changes in the blood that do the mischief. The hæmoglobin cannot in most cases be accurately measured, as the instrument of v. Fleischl, which is the only one available for clinical work, is very inaccurate for percentages of 20 or less. It is very difficult, therefore, to make sure whether the relative increase in hæmoglobin (a larger amount per corpuscle than the normal), which is commonly attributed to this disease, is genuine or not. In our cases it has seemed to be present whenever we could trust the reading of the v. Fleischl instrument.[3]

The white corpuscles are usually diminished in number, sometimes very much so; occasionally they are slightly increased. The blood plaques are much diminished.

Variations in the shape of the corpuscles (poikilocytosis) sometimes occur, but are never more marked than in some cases of secondary anæmia or severe chlorosis, and are not infrequently absent. In 28 cases examined by us 8 showed little or no variations in the shape of the corpuscles, and 5 no variations in either shape or size. Some idea of the appearance of the commoner deformities seen in this disease or in any severe anæmia may be obtained from Plate IX., Fig. C. In some cases these deformities come and go at different periods of the disease, but their presence or absence has, to our knowledge, no definite meaning.

Of more importance is the increase in the average diameter of the red corpuscles, which was present in three fourths of the cases exam-

[1] Hale White, *Brit. Med. Journ.*, 1893, p. 699.

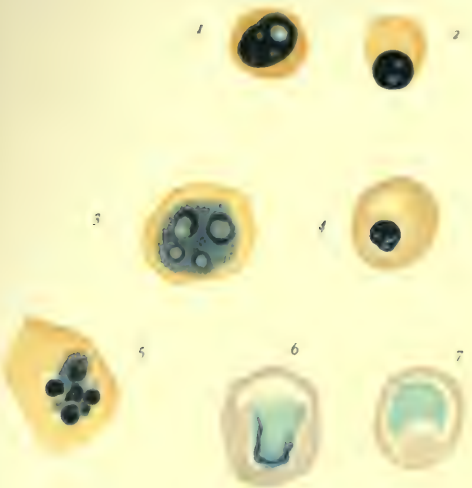
[2] Koettnitz, *Berl. klin. Woch.*, 1890, No. 35.

[3] To avoid this difficulty we may use two pipettesful of blood, and divide the reading by 2.

PLATE IX.

Varieties of Nucleated Red Corpuscles.

FIG. A.



1 and 2 Normoblasts.

In (*1*) note light spots on Nucleus.
In (*2*) note that the Nucleus is protruding from the Cell.
The Nucleus is always deeply stained and may be in process of division.

3, 4, 5, 6 and 7, Megaloblasts.

Cell body always larger than normal (*i. e.*, over 10 μ in diameter). Nucleus usually large, often pale stained and generally surrounded by a white ring. The large size of the cell is its only distinguishing mark. Its protoplasm often shows degenerative changes (see Nos. *4* and *6*), *i. e.*, is polychromatophilic.

FIG. B.

Normal red corpuscles stained with Ehrlick's mixture.

(*6 to 9 μ in diameter.*)

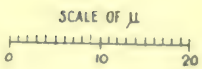
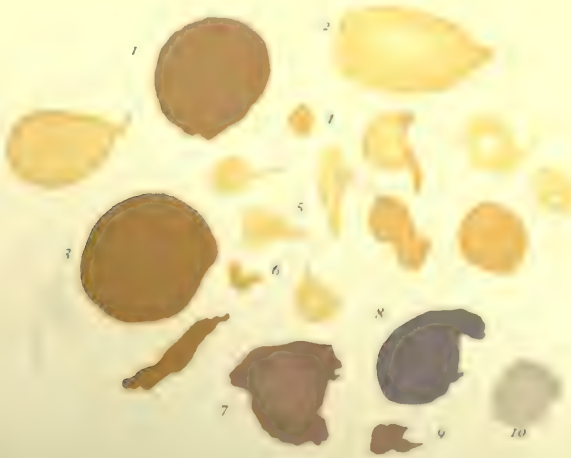


FIG. C.



Degenerative Forms. *1*, *2* and *3*, Macrocytes; *4*, *5* and *6*, Microcytes; *7*, *8*, *9* and *10*, Polychromatophilic forms.

All are misshapen (=Poikilocytes).

ined by us, so far as could be judged without measuring any considerable number of cells. Some of the corpuscles in well marked cases reach a larger size than is to be seen in any other diseased condition, and in 1 case we measured a red cell 17 by 19.6μ, the average normal being 7.5μ. In secondary anæmia or chlorosis one does not find so many cells over the normal size, and never such giants as the one just mentioned.

Nucleated Red Corpuscles.—The most important characteristic of the blood of pernicious anæmia is the presence of *certain types* of nucleated red cells. Nucleated corpuscles are usually divided into two groups:

(1) *Normoblasts* (Plate IX. Fig. A), in which the cell body is of normal or approximately normal size, and the nucleus is round, deeply stained, excentrically placed, and frequently protruding to a greater or less extent from, or even lying by the side of, its corpuscle. These cells are found in all severe secondary anæmias, in chlorosis and leucæmia, and sometimes in the first few days of the life of healthy infants, as well as in pernicious anæmia, so that their presence even in large numbers is in no way distinctive of the latter disease.

(2) *Megaloblasts* are cells considerably larger than the normal and possessing a large, faintly-staining nucleus. (Plate IX. Fig. A.)

It is the presence of a majority of these latter cells over the number of normoblasts present that is characteristic of pernicious anæmia. Megaloblasts are occasionally to be seen in the severest cases of secondary anæmia, but, so far as we are aware, have never been found in numbers greater than the number of normoblasts, except in pernicious anæmia. It is not upon the actual number of megaloblasts, but on their number relatively to the normoblasts, that the diagnosis of pernicious anæmia rests.

So far, the doctrine is very clear, and in some cases we find only these two types of nucleated red cells; but there are in a majority of cases cells which are different from either of these types. We find nuclei of the normoblastic type in a cell body of the megaloblastic type, or the large pale nucleus of the megaloblast nearly filling a normal sized cell. We find cells both large and small whose dark nuclei are in process of division; cells otherwise resembling a megaloblast, but whose nuclei stain intensely dark; small dark nuclei with shreds of protoplasm clinging round them (microblasts); and various other forms.

It is our impression (based, unfortunately, on a small number of cases) that all forms of nucleated red corpuscles, except the normoblast (as above described) and the small cells with dividing nuclei, carry with them a bad prognosis wherever they exceed the number of normoblasts. Most of the reported cases make no distinctions between the nucleated red corpuscles beyond those of normoblast and megaloblast, and the occurrence of these other forms is not discussed as fully as it should be.

The number of nucleated corpuscles varies very suddenly and very widely from time to time in pernicious anæmia. One week we may find only five or six in a cover-glass, and the next week two or three hundred in the same space. As a rule, they become more frequent as death approaches, but this is not always the case. Neither does a sudden and great increase in their number always bring with it any improvement in the symptoms, as has been sometimes supposed. Nucleated

corpuscles were entirely absent in one case (with autopsy) observed by us, and in another (also with autopsy) only a single one was found after prolonged search—a large cell with dividing nucleus. In the other 47 examinations there was always a minority of normoblasts and a majority of other forms of nucleated red corpuscles. Cells whose nucleus showed karyokinetic figures occurred in four of our cases.

Not infrequently the protoplasm about the nucleus of a megaloblast takes with Ehrlich's stain the same dull purplish or pinkish color as in the mononuclear leucocyte, instead of the orange yellow which normal red corpuscles take up with this stain. When this is the case it may be almost impossible to be sure whether you are dealing with a red or a white corpuscle. This same purplish or brownish color is also taken up by some non-nucleated red corpuscles in cases of pernicious anæmia, the protoplasm having undergone some change which gives it an affinity for the basic element in the tri-color mixture as well as for the acid stain which it usually prefers. (See Plate IX., Fig. B.) Hence they are known as *polychromatophilic* cells. These cells are supposed by Ehrlich to be degenerative forms, by others to be "half-baked" or regenerative forms. Whatever their nature, they appear to be more common in pernicious anæmia than in secondary anæmias or in chlorosis, and help to make up the blood picture of this disease. Such cells usually show no signs of the biconcavity of normal red cells, and are generally otherwise misshapen.

Motility in the projecting ends of deformed red corpuscles has been repeatedly observed, and has led to the mistaken reports of motile organisms in the blood. It is due to irregular contractions of the necrobiotic protoplasm.[1]

The whole number of *leucocytes* in pernicious anæmia are usually diminished, as above noted. The small mononuclear forms are relatively increased, sometimes rising as high as 70 per cent. In two thirds of our examinations they were over 30 per cent. This is in sharp contrast with most cases of secondary anæmia where the small mononuclear forms are diminished and the polymorphonuclear cells increased. In chlorosis, on the other hand, and in the many cases of anæmia occurring in syphilis and rickets, we find the same increase in the lymphocytes as in pernicious anæmia.

A more peculiar feature of the leucocytes in pernicious anæmia is the occurrence of small percentages of *myelocytes* (the cells found in such large percentages in splenic myelogenous leucæmia). Of the last 28 cases examined by us, 24 showed myelocytes; 2.6 being the average number present in every 100 leucocytes. They may run as high as 10 per cent. This, indeed, is not peculiar to pernicious anæmia, for myelocytes are found also in chlorosis, secondary anæmias, and various other diseased conditions; but, so far as we are aware, their presence is more common in pernicious anæmia than in any other disease except leucæmia. As a rule, the larger forms of myelocytes are those found in pernicious anæmia, there being less variation in size among them than in leucæmia.

To sum up, the salient points in the blood of pernicious anæmia are—

[1] Solutions used for diluting the blood before counting frequently become the breeding place of various motile organisms, which may lead to error.

1. A reduction of the red cell to the neighborhood of 1,000,000 or less.

2. The absence of leucocytosis, the white cells being usually diminished.

3. In some cases a relatively high percentage of hæmoglobin.

4. The presence of nucleated red corpuscles, of which a minority are normoblasts and the majority either megaloblasts or atypical forms.

5. The presence of polychromatophilic red corpuscles.

6. An increase in the average diameter of the red cells.

7. An increased percentage of lymphocytes, with corresponding decrease in the polymorphonuclear cells.

8. The presence of small percentages of myelocytes.

Duration and Course.—The average duration of a case of pernicious anæmia in this country is from one to three years, rarely more. The disease is rarely progressive, for it is a very constant feature of such cases to show one or more periods of marked improvement in all the symptoms, with or without treatment, followed by relapse in spite of any treatment in use up to the present time. In these remissions the blood count may rise to normal, and the patient go about his work for a few weeks or even for months; but it is very important at such times to be prepared for the inevitable relapse, and not to raise false hopes of recovery. In one case observed by us, in which all drugs had been omitted on account of persistent diarrhœa, the blood count began to rise and the symptoms to improve, and continued to do so until the patient left the hospital feeling perfectly well, only to relapse and die two months later.

As a rule, the remissions occur without any relation to the treatment, which is equally powerless to do more than somewhat delay the subsequent relapse. Of our 66 cases, 59 showed one or more of these unaccountable remissions and relapses.

Diagnosis.—To exclude secondary anæmia is the principal point. As a rule, the blood examination suffices. A secondary anæmia sufficient to reduce the red corpuscles to the vicinity of 1,000,000 almost invariably shows a marked leucocytosis, as well as great emaciation, and usually some distinctive physical sign or group of symptoms. The quality of the pallor is often distinctive, and the presence of retinal hemorrhages is very characteristic. The symptoms pointing to diseases of the spinal cord are very suggestive of the pernicious character of any case of anæmia.

In doubtful cases the finer points of the blood examination may become of paramount importance, the presence of nucleated red corpuscles, of which a minority are normoblasts, being the feature most to be relied upon. In children it may be very difficult to distinguish the disease from leucæmia (p. 701).

Chlorosis shows always a low color index (*i. e.* the hæmoglobin per corpuscle is more diminished than the number of corpuscles), and the normoblasts predominate over the megaloblasts in case nucleated red cells are present.

Prognosis and Treatment.—In this country and with our present poverty of therapeutic resource the outlook is hopeless. In some cases the use of arsenic, in doses increased up to the limit of toleration

and continued over long periods, seems to delay the progress of the disease. But as spontaneous waves of improvement are so marked a feature of the disease, it is hard to be sure that arsenical treatment does anything more than coincide with or possibly increase the power of one of these waves.

We have failed to get any good results from the intestinal antiseptics recommended by Hunter on theoretical grounds or from the massage treatment advised by Mitchell. In 3 cases in which this last was tried there was not even any temporary increase in the corpuscular richness of the peripheral blood. The use of bone marrow has been enthusiastically praised, but in the supposed successful cases we lack one or both of the data mentioned above as indispensable where the word "*cure*" is to be used. It has been of no avail in the 4 cases in which we have watched its effect, using the fresh, uncooked beef marrow spread on bread.

The maximum of nutrition by stomach and bowel should be aimed at, and the symptoms alleviated. The arsenical treatment had best be tried, using Fowler's solution, two drops thrice daily, and increasing one drop daily until warned to desist by toxic symptoms (itching or swelling about the eyes, nausea or diarrhœa, pain or tenderness along nerve trunks). After a pause of a few days the arsenic can usually be resumed, beginning with one half the highest dose previously given. Iron appears to be of no service.

The use of oxygen coincided with a very notable improvement in one of our cases. Whether this is pure coincidence or not we do not feel sure.

(*c*) Secondary Anæmia.

The commonest morbid conditions to which we consider anæmia as secondary are the following:

1. *Hemorrhage*, as from gastric ulcer, menorrhagia and metrorrhagia, hemorrhoids, hæmophilia, and traumatism.

2. *Poisoning*, especially from lead and arsenic.

3. *Infectious diseases*, especially malaria, syphilis, typhoid.

4. "Prolonged drain on the albuminous materials of the blood,"[1] including Bright's disease, chronic diarrhœas, chronic suppurations, and cirrhotic liver.

5. *Malignant disease:* Cancer of the gullet starves the patient into anæmia. How other forms of malignant disease produce the anæmia is unknown.

6. *The anæmias of insanity*[2] are so frequent and so pronounced that it seems as if, either as effect or cause, they must have some essential connection with the mental derangement.

Neusser's[3] discovery of qualitative changes in hysteria are interesting in this connection.

Very severe anæmia in connection with the later stages of *leprosy* have been studied lately by Winiarski.[4]

[1] Osler, *American Text-book of Medicine*, vol. ii. p. 210.

[2] Houston, *Boston Med. and Surg. Journal*, Jan. 11, 1894; Smith, *Journal of Med. Science*, Oct., 1890; Roncoroni, *Revue neurologique*, 1894, No. 11.

[3] *Loc. cit.*

[4] *Petersburg med. Woch.*, 1892, No. 17.

The Blood.—Probably the commonest examples of this condition are found in the course of tubercular or malignant disease, and in a very considerable proportion of these cases the blood has the constitution sometimes supposed to be peculiar to chlorosis[1]—that is, it is the hæmoglobin rather than the number of corpuscles that suffers. It is not at all uncommon in the blood of pale-faced consumptives or cancer patients to find over 4,000,000 red cells, or even over 5,000,000 (the influence of diarrhœa or venous stasis in concentrating the blood must of course be allowed for). In such cases we are apt to find only from 30 per cent. to 50 per cent. of hæmoglobin.[2] Thus in 48 cases of cancer and 20 cases of tuberculosis which ran their course without leucocytosis under our observation the averages were as follows: red cells—cancer, 4,100,000; tuberculosis, 4,400,000; hæmoglobin—cancer, 52 per cent.; tuberculosis, 56 per cent. The same is true to a lesser extent in most other secondary anæmias, especially those due to Bright's disease and cirrhosis of the liver. In the anæmia following hemorrhage, while we do not so often have the typical "chlorotic" blood, as seen in Bright's disease or tuberculosis, the hæmoglobin usually suffers more than the corpuscles, and in convalescence is slower in reaching the normal.

Leucocytosis is the rule in the anæmias due to rapidly growing tumors, hemorrhage, and prolonged suppurative diseases where the infection is not purely tubercular;[3] occurs occasionally after malaria,[4] syphilis, or cirrhotic liver; is rare in the toxic anæmias from lead, arsenic, and in the course of chronic nephritis. The subject will be more fully discussed under Leucocytosis (p. 691).

The reduction in the red cells is rarely so great as is the rule in pernicious anæmia, although no absolute distinction can be drawn by the count alone. Nucleated red corpuscles in small numbers are the rule in severe cases, especially in malignant disease, and are mostly but not exclusively of the normoblastic type, megaloblasts also occurring.

Irregularity in size and shape of corpuscles is common in all the severer grades of anæmia, and may be as great as in pernicious anæmia. Strauer observed it much more frequently in malignant disease than in tuberculosis.

Polychromatophilic red corpuscles also occur, though less frequently than in pernicious anæmia.

The lack of albuminous constituents and the lowering of the specific gravity are sufficiently well measured by the hæmoglobin percentage with which they both run parallel.[5]

Symptoms.—The pallor is usually distinctly different from the peculiar yellow of pernicious anæmia (though sometimes the two have exactly the same color), and also from the color of chlorosis. In cirrhosis of the liver and chronic malarial poisoning there is sometimes the "muddy pallor" so often spoken of, but in lead-poisoning and in some cases of malignant disease there is nothing to distinguish the color from that of pernicious anæmia.

[1] Laker, *Wien. med. Woch.*, 1886, 18 and 19.
[2] Häberlin, *Münch. med. Woch.*, 1888, No. 22.
[3] Dane, *American Journal Medical Sciences*, 1896.
[4] Billings, *Johns Hopkins Hosp. Bull.*, 1894, No. 46.
[5] Strauer, *loc. cit.*

The symptoms common to all anæmias, such as muscular weakness, dyspnœa, vertigo, syncope, anorexia, vomiting, etc. (p. 663), are usually present to a greater or less extent. An interesting feature of the acute anæmias secondary to hemorrhage is the occasional presence of high and continued fever. One of our cases, which followed a large hæmatemesis, was a gastric ulcer, had a temperature of 103° for three days continuously, although the patient felt well and was not much exhausted. She was very neurotic, and it is specially in this type of person that fever after hemorrhage is to be seen.

There is usually emaciation contrasting with the well preserved fat layer of chlorosis and of most pernicious cases of anæmia. In other respects the symptoms of secondary anæmia are usually masked by those of the diseases to which it is secondary and hardly to be separated from them.

The PROGNOSIS depends, of course, largely upon the underlying disease. If the cause can be removed, it is, as a rule, favorable. Long standing cases, such as occur from chronic malaria or lead-poisoning, are naturally more resistant to treatment.

TREATMENT.—Removal of the cause is of course the first indication. Beyond that, the maximum of nutrition, sometimes using the rectum as well as the stomach, hydrotherapy, iron (given as in chlorosis, p. 666), and arsenic (given as in pernicious anemia, p. 676), are our principal agents. Massage has been recommended, its action being probably, like that of hydrotherapy, through the nervous system.

LEUCÆMIA; LEUCOCYTOSIS; HODGKIN'S DISEASE; THE BLOOD IN INFANCY.

By FREDERICK C. SHATTUCK, M. D., and RICHARD C. CABOT, M. D.

LEUCÆMIA.

We may distinguish two types of the disease: (*a*) the splenic-myelogenous, and (*b*) the lymphatic, according as the disease affects chiefly the lymphatic glands or chiefly the spleen and marrow. As a rule, all three sets of organs are somewhat affected, but unequally. Pure splenic and pure myelogenous leucæmia are so rare that their existence may be doubted.[1]

(*a*) *The splenic-myelogenous form* is characterized by the presence in the blood of enormous numbers of cells apparently originating in the marrow, and called by Ehrlich "myelocytes;" by a very great increase in the size of the spleen with relatively little enlargement of lymphatic glands; and in most cases by a relatively chronic course.

(*b*) *The lymphatic form* shows in the blood a greatly increased percentage of the small mononuclear leucocytes relatively to the other white cells present (the whole number of leucocytes being also greatly increased), together with enlargement of the lymphatic glands in various parts of the body. It tends to run a much shorter course than the splenic-myelogenous type, although chronic cases also occur.[2]

The great majority of cases can be grouped under one of these two headings, but mixed forms also occur where some characteristics of each type are present.

Etiology.—The etiology of the disease, like that of pernicious anæmia, is entirely unknown. Various micro-organisms have been described by various writers in connection with it,[3] but negative results are also plenty,[4] and the organisms where present are probably to be accounted for as a sign of that decreased resistance to bacterial invasion which has been observed toward the close of other diseases. Nevertheless, there is a good deal of evidence in favor of there being some infection closely associated, at any rate with many cases. The acute febrile course of some cases, and especially the unique observations of Obrastow,[5] cannot be left out of consideration. A ward-tender who had

[1] The reported cases are—(*a*) Neumann, *Berl. klin. Woch.*, 1878, Nos. 6–10; (*b*) Leube and Fleischer, *Virchow's Archiv*, vol. lxxxiii; (*c*) Beatty, *Roy. Acad. of Med.*, May, 1891; (*d*) Eichhorst, *Virchow's Arch.*, vol. cxxx.

[2] Hinterberger and Fränkel, *Deut. Arch. f. klin. Med.*, xlviii. p. 338.

[3] Pawlowsky, *Deut. med. Woch.*, 1892, No. 28, p. 341.

[4] Elstein, *Deut. Arch. f. klin. Med.*, 1889, p. 343.

[5] *Deut. med. Woch.* 1892, p. 641.

charge of a patient suffering from acute leucæmia under Obrastow's care, who had attended to the patient's urine and fæces, and assisted in examining the blood and in plugging the nostrils for profuse hemorrhages, was also seized, forty days later, with acute leucæmia, and both cases ran a similar course and died within a short time of each other. Both were clearly leucæmia of the lymphatic form, and evidently acute, the blood being normal in both cases a few weeks before death. There was an interval of forty days between the end of the first case and the beginning of the second, but in all other respects the resemblance to contagion was striking. No one has yet confirmed the observations of Pawlowsky,[1] who reported a certain bacillus in six successive cases.

A great deal of stress has been laid upon the association of ulcerations in the mouth or in the intestine in connection with most of a group of acute cases collected by Hinterberger.[2] The ulcerated surface has been thought to be the point of entrance of the *materies morbi*, and the same has been inferred from the fact that in some instances a diarrhœa has preceded the other symptoms complained of.

Of the 47 cases known to us, only 1 showed any lesion in the mouth, and that late in the course of disease; and in none was there any history of diarrhœa before the enlargement of the spleen was noticed. Only 2 of these cases were acute, but it does not seem likely that the acute cases have a different etiology from the chronic ones. It appears to us more reasonable to suppose that the course of those cases in which ulcerations occurred was hastened by absorption of some toxic substance through these lesions, themselves a symptom of the disease.

Most chronic cases end with "acute" symptoms resembling those described in the acute leucæmia; the thought of a secondary septicæmia, due to bacterial invasion of the half-dead tissue, inevitably suggests itself, and receives some confirmation from the finding of pyogenic organisms post-mortem in the organs of many cases.

That the disease bears a relationship to pernicious anæmia is to be argued by the rare but well certified instances of transition from the one to the other condition,[3] and is further suggested by the very constant presence of myelocytes in pernicious anæmia (the percentages running as high as 8 to 10 per cent. of the total leucocyte count), as well as by other points of similarity, especially when the disease occurs in children. The relationship to Hodgkin's disease must also be very close, since the pathology of the two is identical, and transitions from Hodgkin's disease to true leucæmia have been observed.[4]

In 5 cases of Hodgkin's disease we have found small percentages of "myelocytes," 2.6 per cent. being the largest number.

On the whole, we are inclined to regard leucæmia as more akin to certain forms of malignant disease (*e. g.* acute sarcomatosis) than to a bacterial infection.

The occurrence of 28 cases within three years in our own personal experience, all from the vicinity of Boston, shows that the disease is not a very rare one.

Country, race, and season seem to be of no importance in the causation of the disease.

[1] *Loc. cit.* [2] *Loc. cit.* [3] Laache, *Die Anämie*, Christiana, 1883.
[4] Palma (*Deut. med. Woch.*, Sept. 1, 1892) refers to 5 such cases, besides 1 of his own.

Males are more subject to it than females, 67 per cent. of Birch-Hirschfeld's cases being in males; there were 29 males out of the 47 cases known to us. Like pernicious anæmia, it usually occurs in middle life, although it may come at any age.[1] Only 3 of our cases came from the well-to-do classes—a fact in strong contrast with our experience as to pernicious anæmia. In other statistics the difference seems to be less marked.

Malaria, syphilis, pregnancy, parturition and the climacteric, mental strain, and traumatism have been supposed to have causal connection with leucæmia as with pernicious anæmia. We believe them to have as little significance in the one case as in the other, and to act only as predisposing elements if at all. Only 1 of our 18 female cases had passed the climacteric. In 2 the disease was first noticed soon after parturition. Trauma[2] seems in some cases to have intensified the previously mild symptoms, and the acute downward course of a chronic case sometimes dates from a fall or strain. Heredity seems to play a certain part in the etiology (as with malignant disease), but leucæmic women have borne healthy children. The disease is so much aggravated during pregnancy that the question of emptying the uterus in the interests of the mother has sometimes to be considered. Of the two forms of leucæmia, the splenic myelogenous is by far the more common. Of our 47 cases, only 8 were of the lymphatic type.

Symptoms.—The disease may be acute or chronic. The majority of sufferers from chronic leucæmia are in very fair general condition when first they consult the physician (or often the surgeon) about the "lump" in the abdomen. Not infrequently patients are not even aware of the splenic enlargement, which is first discovered in course of routine physical examination by the physician to whom they come for some trifling gastric or respiratory disorder. It forms a marked contrast in this respect with pernicious anæmia, where the general condition is usually bad when the physician is first called. Three cases under our observation have been able to attend to light work for from one to two years, and 1 case expressed herself at the end of two years as feeling perfectly well—said she did whatever she liked and could walk "any distance." At this time her spleen filled over one half the abdomen, and a cubic centimetre of her blood contained 320,000 white corpuscles. She was rosy and plump. Another patient who had had the disease at least one and a half years passed safely through a severe attack of croupous pneumonia, and for a time went on earning her living by washing.

Sooner or later there gets to be some pain or discomfort over the spleen, and the gradually increasing debility and anæmia disable the sufferer. Gastric symptoms are not very common, and the appetite may remain good to the end. Diarrhœa was a marked symptom in one sixth of our cases, and is much to be dreaded, as it is often unaffected by drugs, and stops of itself if at all, as is the case in pernicious anæmia. The movements are occasionally bloody, but there is nothing distinctive about them. The bowels are sometimes costive from the pres-

[1] Compare Vehsemeyer, *Münch. med. Woch.*, Oct. 23, 1894.
[2] Ebstein, *Deut. med. Woch.*, July 26, 1894.

sure of the enlarged spleen, and actual obstruction may occur. They were regular in two thirds of our cases.

Circulatory symptoms are more marked, for the heart is pushed up and its action embarrassed, sometimes very severely, by the growing spleen. Ascites and œdema of the legs are frequent symptoms, and hydro-thorax is not rare.

Shortness of breath on over-exertion is common, owing to the combined influence of the anæmia and the embarrassed heart action.

Hemorrhages are not uncommon, occurring in one third of our cases. The nose is the most frequent seat of bleeding; fatal nosebleed occurred once in our series, and fatal cerebral hemorrhage once. Other seats of hemorrhage are the brain, the gums, the subcutaneous tissues, the stomach, and the bladder. One of our cases died suddenly of cerebral hemorrhage, having felt practically well till a few hours before his death. Hemorrhages are much more common in acute lymphatic leucæmia, occurring in almost all the recorded cases, especially from the gums, and the foulness of the mouth in such cases is almost incredible.

The pulling of a tooth started a severe hemorrhage in one of our cases, but in general there is no true hæmophilia.

There may be dyspnœa or dysphagia from the pressure of enlarged glands on the gullet or windpipe, but pressure symptoms are much less common than in Hodgkin's disease.

Deafness is not uncommon, owing to the growth of leucæmic nodules in the internal ear. It came on suddenly in 4 of our cases.[1]

Defects of eyesight are also noticed by the patients occasionally.

Toward the close of the disease, if it be not interrupted by some intercurrent infection, weakness rapidly increases, dyspnœa and cough from the anæmia and œdematous lungs supervenes, the emaciation becomes extreme, and the patient dies of exhaustion.

In several of our cases there has been from time to time a severe rigor with sharp rise of temperature to 103° or 104° F., vomiting, and sweating. No cause whatever has been discovered, and we have supposed the symptoms to be due to the entrance of some poison or organism against which the system was not strong enough in its weakened state to protect itself. We have seen similar unaccountable chills in pernicious anæmia, in patients in the fourth and fifth week of typhoid, and in malignant disease, and supposed the explanation to be the same.

Physical Examination.—Most patients are rather pale, but flushed cheeks are sometimes seen, though the lips may be white. In many cases the color is not such as to attract notice, and some are fairly ruddy.[2] There may be a distinct brownish yellow discoloration to the skin, much darker than that of pernicious anæmia. This was present in 5 of our cases previous to the use of arsenic. In 1 case it seemed to follow the use of the drug. Purpuric spots old and new are occasionally to be seen, and in the acute form of the disease hemorrhages from the skin and mucous membranes are an early and prominent sign.

Enlarged glands are sometimes to be felt in the neck, submaxillary and occipital regions, as well as in the groin and axilla and along the

[1] Lamois (*Lyon méd.*, Jan. 3, 1892) noticed a vertigo like that of Ménière's disease in a case of leucæmia.

[2] Schultze, *Deut. Arch. f. klin. Med.*, vol. liii.

pectorals, even in the splenic-myelogenous form. In the lymphatic form the glandular enlargement is much greater, but the glands never attain the size commonly seen in Hodgkin's disease, and, except in the most acute cases, there is no tendency to suppuration in them. The skin is sometimes involved; nodular elevations appear and may ulcerate. Such cases have been called *lymphodermia perniciosa*.

The apex of the heart is usually pushed up into the fourth interspace, and its action may be rapid and irregular. If much anæmia is

FIG. 47.

Outline of the spleen in splenic-myelogenous leucæmia (chronic). The outline of the subcostal portion is shown by a dotted line. The patient lived thirteen months from the time when this photograph was taken.

present, we get the systolic murmurs common in that condition. The heart dulness often merges into that of the enlarged spleen, which grows upward as well as downward, although by far the larger portion of it is in the abdomen below the rib margin.

The lungs show no special signs except the moist râles of œdema at the base in advanced cases.

The spleen usually fills at least one half the abdomen, forming a firm, smooth, flat tumor immediately beneath the abdominal wall, so that its edge can often be taken in the hand. It is usually not tender. In the edge nearest the navel one or two deep notches can usually be

felt, rarely three. It is dull or flat on percussion, but can be so easily felt that percussion is of value chiefly in indicating the outline of the subcostal portion (Fig. 47).

In the lymphatic form of the disease the spleen usually projects only from one to three fingers' breadth below the ribs (see Fig. 48), but in

FIG. 48.

Outline of the spleen in lymphatic leucæmia (chronic): the subcostal portion indicated by a dotted line. Patient lived twenty-two months from the time when this photograph was taken. Lymphatic glands much enlarged.

rare cases it may reach the navel.[1] In the other and much commoner form of the disease it may reach to the right iliac fossa and touch the iliac crest on both sides. The direction of its growth is obliquely downward and to the right. Under treatment or during intercurrent infections it may recede almost to the rib margin. In acute cases it some-

[1] Eichhorst, *Virchow's Arch.*, vol. cxxx.

times grows much softer toward the end. A friction rub, due to a perisplenitis, can sometimes be heard or felt, and a systolic souffle has been heard over the spleen.

The liver is usually enlarged. In one third of our cases it reached the navel, and its firm, smooth edge could be felt and traced to where it meets the edge of the spleen. It also grows upward, often as high as the fourth interspace. A certain amount of ascites is usually present, and the legs are frequently œdematous.

Examination of the retinæ shows hemorrhage to be fairly common there, although less so than in pernicious anæmia. Sometimes there are also white streaks and spots due to masses of lymphoid growth, to which the name of "leucæmic retinitis" has been given, and the optic nerve is not infrequently inflamed.

As before said, we may find subcutaneous nodules from the size of a pea to that of a walnut, and the disease may first be discovered by the dermatologist.

The external ear shows nothing but pallor of the drum membrane even where complete deafness is present, the changes being wholly within. Sukarinek[1] has noted a nasal growth of leucæmic origin.

Nodules may sometimes be seen in the throat with the laryngoscope.

Tenderness over the long bones is not a common symptom, and occurs in various other non-leucæmic conditions.[2] It was present in only one tenth of our cases.

The urine shows nothing pathological except an excess of uric acid and xanthin bodies, and sometimes the evidence of passive congestion of the kidneys.

Fever is the rule, ranging in chronic cases between 99° and 100.5° as a rule; in acute cases higher.

Mental dulness is common in the acute form of the disease and toward the end of all cases, and there may be delirium. Facial paralysis from leucæmic infiltration of peripheral nerves has been recorded by May, and bulbar paralysis has occurred.

Priapism, persisting for days or even weeks, occasionally occurs. It is not due to hemorrhage into the corpora cavernosa, as has been stated, and the cause is unknown.

The Blood.—A drop of the blood usually looks entirely normal or at most somewhat pale. The whitish, brownish, or chocolate color mentioned by some writers was not present in any of our cases; probably the appearances of the large post-mortem clots seen at autopsy have accidentally crept into the descriptions of the blood as seen at the clinical examination. The notable change in gross is the decreased fluidity and sluggish, heavy flow of the drop, which makes it very hard to spread between cover-slips. Rarely there is a hemorrhagic tendency.

The red corpuscles are usually diminished in advanced cases, averaging 3,000,000 in our cases, but we have seen five well-marked cases with over 4,500,000 red cells per c.mm. Early cases show no anæmia.

It is chiefly by the blood that the two types of the disease are to be distinguished. In the splenic-myelogenous form the increase of white cells is usually greater than is recorded in any other disease. No pure leucocytosis (*i. e.* one where the proportion of polymorphonuclear cells

[1] *Arch. of Otology*, 1891, vol. xix. p. 4.

[2] Schultze, *loc. cit.*

is increased) of over 150,000 cells per c.mm. has ever been reported,[1] while the average count of white cells in our leucæmic cases was 350,000 at the time they came under observation.

In the lymphatic form the increase of white cells is usually not so large—not larger than many leucocytoses, from which it can only be distinguished by staining the blood. A proportion of 1 white to 20 or even 40 red is the rule. The average ratio in our 8 cases was 1 : 27, while in 39 splenic-myelogenous cases it was 1 : 8.

The hæmoglobin is usually diminished, sometimes proportionally with the count of corpuscles, sometimes more decidedly. There is nothing distinctive about it (Plate X.).

Stained Specimens.—I. *Splenic-myelogenous Form.*—Stained with Erlich's tri-color mixture and examined with a $\frac{1}{12}$ immersion lens, the field is full of large, round, pale-staining corpuscles, whose protoplasm is seen (when properly stained) to be crowded with fine pinkish or brownish granules visible chiefly at the periphery. The nucleus fills most of the cell, is pale purplish or greenish blue, and very evenly colored throughout, except for an occasional vacuole. The nucleus is round or oval, and is in contact with the surface of the cell for a considerable portion of its extent, usually lying excentrically. These cells are the "*myelocytes*" of Ehrlich. They are *usually* larger than the other white cells in the field, but they vary from 8μ to 23μ in diameter. These cells are not peculiar to leucæmia, for, as mentioned above, we may have considerable percentages of them in pernicious anæmia, and they are occasionally found in many other conditions. But the number of them present in the blood is enormously greater in leucæmia than in any other condition. Thus the largest number of myelocytes yet reported in pernicious anæmia is 9.2 per cent.[2] The leucocyte count being 8000, this gives less than 800 myelocytes per cubic millimetre, while in leucæmia, with the average number of white cells present in our cases, 9.2 per cent. of myelocytes would mean 25,000 per cubic millimetre. But in untreated leucæmia we have never seen the percentage of myelocytes so low as 9.2 per cent. The average percentage in our cases was 37, or about 80,000 per cubic millimetre. The number of myelocytes, therefore, averages at least one hundred times greater in leucæmia than in pernicious anæmia or any other disease.

Under treatment, as Osler[3] points out, the count of leucocytes may fall to normal, the myelocytes still remaining present in small numbers. Under these circumstances the blood might be undistinguishable, so far as the leucocytes are concerned, from that of pernicious anæmia. The lower count of red cells and the larger proportion of megaloblasts in the latter disease would be our best distinguishing mark.

The *polymorphonuclear* cells are relatively much diminished, although the absolute number is, of course, greatly increased. They are mostly of very small size as compared with those of normal blood, but even more striking is the *variation* in the size, some being almost

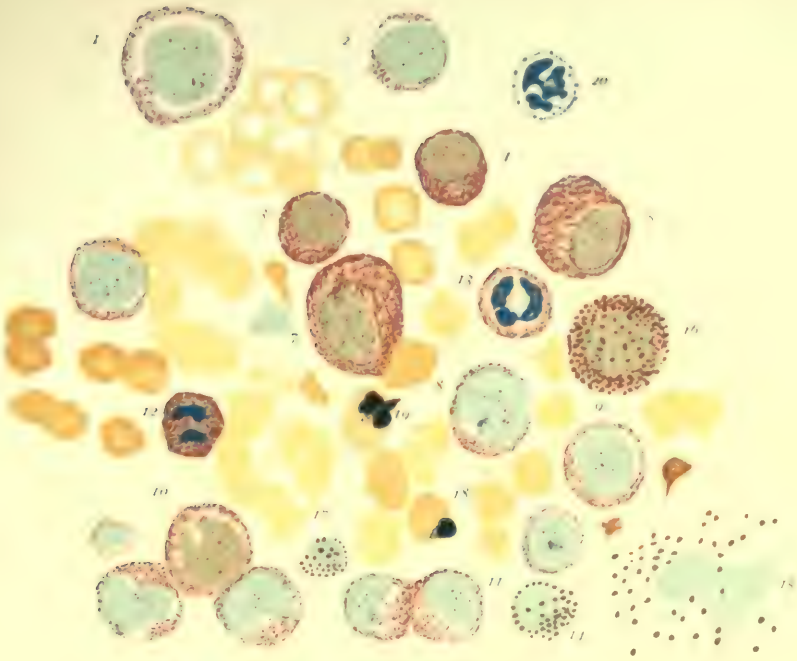
[1] Except in the bubonic plague, where a leucocytosis of 200,000 is referred to by Aoyoma, a Japanese observer: *Mittheilungen aus d. Med. Fac. d. Kaiserlich Japanischen Universität*, vol. iii. No. 1, Tokyo, Japan, 1895.

[2] Cabot, *Boston Med. and Surg. Journal*, Jan. 2, 1896.

[3] *Loc. cit.*, p. 224, vol. ii.

PLATE X.

FIG. A.

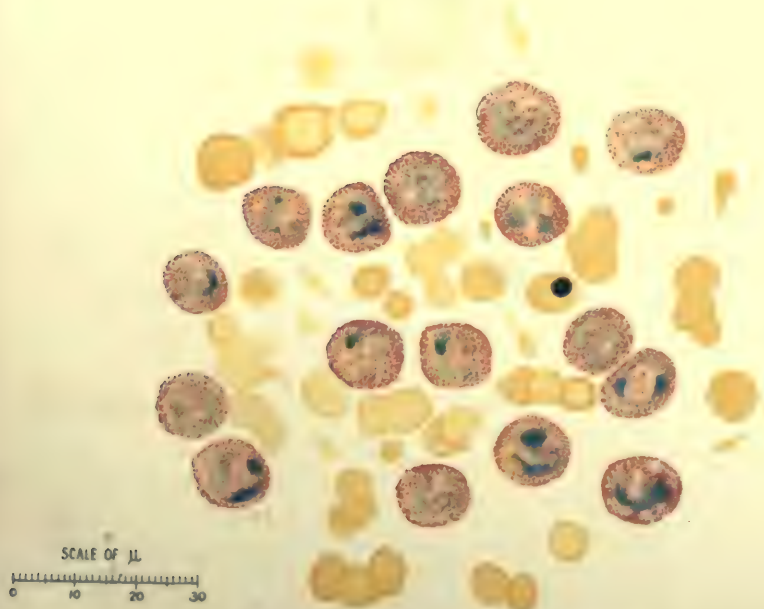


SPLENIC-MYELOGENOUS LEUCÆMIA.

Nos. 1–11, Myelocytes; 12 and 13, Polymorphonuclear Neutrophiles; 14 and 15, Eosinophiles; 16, Eosinophilic Myelocyte; 17, Eosinophilic Dwarf-cell; 18 and 19, Normoblasts (nucleated red cells); 20, "Mast cell."

The red corpuscles show moderate deformities in size and shape.

FIG. B



LEUCOCYTOSIS (Cancer of Kidney).

All the Leucocytes figured are Polymorphonuclear Neutrophiles (=96 per cent. of all in this case).

The red cells much deformed, showing severe anæmia.

as large as the largest myelocyte. The average diameter is about 9μ as compared with 13.5μ in health.

Lymphocytes and large mononuclear cells are hardly to be found, averaging only 6 per cent. and 2 per cent. respectively in our cases.

The percentage of *eosinophiles* is sometimes slightly increased, but was never much so in our cases, averaging 4.2 per cent. It was the absolute and not the relative increase that Ehrlich has pointed out.[1] This is in no way characteristic of leucæmia, though many text-books continue to repeat this long-exploded fallacy.[2]

There are always a certain number of atypical forms of white cells present, such as polymorphonuclear cells without granules or with very large granules, and hardly to be distinguished from eosinophiles with the Ehrlich tri-color mixture; also lymphocytes with two nuclei, basophilic cells, etc.

The red cells are usually somewhat deformed, and nucleated corpuscles of the normoblast and "small, dividing" types are commonly present, sometimes in enormous numbers. Megaloblasts are not numerous, except in cases occurring in infancy.

II. *Lymphatic Form* (see Plate VIII.).—In the lymphatic form we find the field full of small lymphocytes about the size of red cells, and with a very narrow layer of pale pink or colorless protoplasm about the blue nucleus. These make up, usually, rarely *over 90 per cent.* of all the white cells present. In 7 out of our 8 cases the percentage of lymphocytes in every 100 white cells was 99.9, 98.6, 97.9, 96.5, 94, 91.8, and 82.5. In 2 cases the lymphocytes were of all sizes, with no sharp line to be drawn between large and small mononuclear. Counting large and small forms together, they made up 98 per cent. of all the white corpuscles present, there being only $\frac{1}{2}$ of 1 per cent. of polymorphonuclear cells present.

A few myelocytes are usually present, but not more than in any other cachectic condition. We have never seen any of the so-called "mixed" forms. Nucleated red corpuscles are much less common than in the splenic-myelogenous form.

PATHOLOGICAL ANATOMY.—(*a*) The *spleen* is of course the principal feature in most cases. It may be a foot and a half long and half as wide, and is usually bound down more or less by the adhesions of chronic perisplenitis. It is usually firm, but occasionally softens down toward the end of the disease in acute cases, owing to hemorrhage into its substance and great increase of pulp. Even rupture may occur. The notches or lobulations are very striking. The cut surface is dark brownish or purple, often more or less spotted with brownish yellow areas of old hemorrhagic infarction. Microscopically, a hyperplasia of all the elements is to be found, occasionally with circumscribed lymphoid nodules of various sizes, as in other organs.

(*b*) The *lymph glands* are hyperplastic in almost all cases, but especially so in the lymphatic form of the disease. In all well marked cases they are much redder than normal on the cut surface, owing to hemorrhage. In both spleen and glands the characteristic change microscop-

[1] Thayer, *Boston Med. and Surg. Journal*, 1893, vol. cxxviii. p. 183.

[2] *E. g.* Gilbert in *Traité de la Médecine*, Paris, 1892; Strümpell, *Text-book of Medicine*, vol. ii. p. 948, 1894.

ically is an infiltration of lymphoid cells—that is, of cells corresponding exactly with the lymphocytes of the circulating blood. Either the small or the large form of lymphocyte may predominate in the lymphoid organs, as in the blood, or we may find all sizes from 5 to 15μ in diameter. The cells contain no granules when stained by Ehrlich's methods.

(*c*) The *marrow* of the long bones may show no changes at all, as occasionally happens in pernicious anæmia. Such changes as it does show are very differently described by different observers, and are apparently not distinctive of the disease. In gross, it may be yellow or semi-fluid (pyoid marrow) or red as in the fœtus (lymphoid marrow). In both, the fat cells are mostly gone; in the pyoid marrow they are replaced by masses of *myelocytes* (see above), while in the lymphoid marrow we have also numerous *nucleated red corpuscles* to which the red color is due. Neither lymphoid nor pyoid marrow is peculiar to or characteristic of leucæmia.

The liver shows usually an infiltration of lymphoid cells, either diffuse or in grayish white masses, contrasted with the surrounding substance.

Similar tumors and infiltrations are sometimes seen in the kidney, thymus and thyroid glands, intestine, and even in the lungs and serous membranes. In the lymphatic form of the disease the bronchial, mediastinal, and mesenteric glands may be much enlarged; and it is chiefly in this form of the disease that nodules in the skin are seen. These show the same more or less hemorrhagic lymphoid tissue found in the various organs.

Diagnosis.—The diagnosis is usually easy, provided the blood is examined at all. The danger is that a case might be considered malaria, malignant disease, rickets, tuberculosis, or syphilis on the evidence of the enlarged spleen or glands, without thinking of the blood at all. There are many cases of the diseases just mentioned in which the diagnosis from leucæmia would be impossible without the blood. The symptoms and signs of leucæmia present nothing whatever that is characteristic or distinctive. The blood should therefore be examined in all cases presenting anæmia and enlarged spleen or glands, and it must not be forgotten that malaria, syphilis, etc. may coexist with leucæmia. Leucæmia and Hodgkin's disease are identical except for the blood. Acute leucæmia may be easily mistaken for scurvy, Werlhof's disease, or acute infectious diseases accompanied by hemorrhage. The blood, however, is distinctive.

The enormous increase of white cells, together with the appearances described in the stained specimen, is sufficient to distinguish the disease, and other physical signs are only confirmatory evidence. So far as is known, the remissions described by Osler under the use of arsenic, in which the count of white cells becomes normal, do not occur except under treatment. Should such be found to occur spontaneously, the disease could not, during such a remission, be distinguished from the splenic form of Hodgkin's disease. The close resemblance to pernicious anæmia, especially in infancy, has already been noted. (See also p. 701.)

A marked leucocytosis may be taken for leucæmia if no differential count is made, but the two are absolutely and invariably different in their effects on the *kind* of leucocyte predominating. (See Plate X.)

Leucocytosis shows chiefly polymorphonuclear cells; leucæmia, myelocytes or lymphocytes. In infancy the diagnosis between pernicious anæmia and leucæmia may be very hard. (See page 702.)

Prognosis and Duration.—The outlook is hopeless and the question is simply one of time. The longest case known to us was entirely unaffected by treatment and was not in a robust person, so that we cannot be entirely governed in prognosis either by the response to treatment or by the degree of constitutional vigor. Still, these data must affect our judgment. The absence of diarrhœa and of high fever is favorable, as is also the ability of the patient to devote himself to his disease. Four years is probably its limit, and most cases die within two years. The lymphatic form is the more rapid, as a rule.

Treatment.—V. Noorden[1] and others have shown that the assimilation of proteids is far below normal in leucæmic patients; accordingly, Vehsemeyer[2] advises a carbohydrate diet with limited proteids.

The use of arsenic in doses increased until toxic symptoms appear, and maintained up to the limit of toleration, seems in some cases to be of service. Under its use the size of the spleen may greatly decrease and all the symptoms improve. In 3 of our cases the leucocytes fell to less than 40,000 per cubic millimetre during the use of arsenic. It is difficult to be sure that this is due to the treatment, since spontaneous remissions are a feature of the disease, though less constantly so than in pernicious anæmia. Otherwise, our treatment is to be symptomatic and hygienic with a view to keeping up the general condition of the patient, as in debility from any other cause. Bone marrow, iron, quinine, electricity, transfusion, and hydro-therapeutics have no considerable influence on the disease.

LEUCOCYTOSIS.

Leucocytosis is an increase in the number of white cells found in the blood as obtained from the peripheral capillaries, without any considerable diminution in the percentage of the polymorphonuclear variety, which percentage is usually much increased. The influence of venous stasis must be excluded.

It is becoming increasingly difficult to frame a definition that includes all cases of leucocytosis, and yet distinguishes the condition clearly from leucæmia. Before the use of Ehrlich's staining methods became general leucocytosis was supposed to be distinguished from leucæmia merely quantitatively, and the latest French text-book[3] still prints this fallacy, according to which counts below 70,000 leucocytes are "leucocytosis," and counts above this number "leucæmia." In fact, counts of over 70,000 are not uncommon in pneumonia, malignant disease, pyæmia, and the anæmias of infancy, while the disease leucæmia under treatment may show at times no increase at all over the normal number of white cells. The difference between leucæmia and leucocy-

[1] *Pathologie der Stoffwechsel*, p. 346.
[2] *Loc cit.*
[3] Gilbert in *Traité de la Médicine*, Paris, 1892.

tosis depends on the kind of cells present, and not simply on their number.[1] (See Plate X.)

It is still an open question whether the phenomenon of leucocytosis is simply a matter of distribution, the white cells being driven or attracted into the peripheral circulation so as to increase the number found there, or whether the whole number of white cells in the blood is really increased by new cell production. In some conditions the increase takes place solely in the polymorphonuclear varieties at the expense of the other forms. In other cases—as, for example, in the digestion leucocytosis (*vide infra*)—the proportion of the different varieties in any one hundred leucocytes remains normal, and there are cases in which the percentage of polymorphonuclear cells is decreased (see p. 694).

Into the mode of production of leucocytosis we shall not enter at all, as no one of the various theories seems to us to have enough evidence to justify its existence. It remains to enumerate, and as far as possible to classify, the conditions in which it occurs, and to indicate what significance it has in diagnosis and prognosis.

The normal number of leucocytes has been stated at from 5000 to 11,000 per cubic millimetre. A count of 10,000 white cells per cubic millimetre cannot be called a leucocytosis, unless as compared with a lower previous count in the same case.

Provisionally and for convenience' sake we may divide leucocytoses into the *physiological* and the *pathological*, although it may be that the method of their production is the same in all cases.

Physiological Leucocytosis.

Under the head of physiological leucocytosis come—

I. The leucocytosis of the newborn.
II. " " of digestion.
III. " " after exercise,[2] massage, electricity, and cold baths.
IV. " " of pregnancy.
V. " " post-partum.
VI. " " of the moribund state.

I. (See Blood in Infancy, p. 700).

II. The leucocytosis of digestion is more marked in the purely carnivorous animals, and in man is seen only after meals rich in proteids. It begins to appear about an hour after food, and reaches its maximum in from two to three and a half hours after food. The increase is rarely more than 6000 cells over the number present before food. It may occur as an addition to the leucocytosis permanently present from other conditions (such as malignant disease) or as an increase over the normal number. Its importance is—(1) That it must be borne in mind in examining the blood for other leucocytoses, for which we should always take the blood just before, during, or just after a meal, or, if possible, before breakfast.

[1] For an opposite view see Troye, *Berl. klin. Woch.*, March 21, 1892.

[2] Schultze, *Deut. Arch. f. klin. Med.*, 1893, p. 234; and Oliver, *Brit. Med. Journal*, June, 1896.

(2) There is some evidence for believing that its presence in any case militates against the diagnosis of gastric cancer; its absence, like absence of HCl, may be due to a variety of causes. Schneyer[1] first called attention to this. Examinations have been made with reference to this point in 14 cases of gastric cancer at the Massachusetts General Hospital. In 12 of these there was no digestion-leucocytosis. In 3 cases of gastric ulcer there was a digestion-leucocytosis.

III. *Leucocytosis from Increased Blood Pressure.*—Oliver[2] has shown that a variety of influences increasing blood pressure concentrate the blood, and thus increase the absolute numbers of cells per c.mm. at the periphery.

Cold baths of short duration add 6000–8000 to the number of white corpuscles found in a cubic millimetre of the peripheral blood. Prolonged cold baths cause a sinking of the number below normal. Hot baths act in the opposite way, first decreasing, but if prolonged increasing, the number of white cells.

Exercise, electricity, and massage increase temporarily both the leucocytes and the red cells in the peripheral circulation in healthy persons and in some diseased conditions.

In pernicious anæmia they have no effect on the blood.

IV. The leucocytosis of pregnancy, like that of digestion, is not constant in all individuals nor at all times, but, as a rule, is present in primiparæ during the last months of pregnancy, with a gradual increase toward the end. It is of about the same degree as the digestion-leucocytosis, not reaching above 14,000 cells per c.mm. until just before parturition. It has rarely any diagnostic value, as it is not often present in the earlier months after conception, and in the later months most of the conditions that simulate pregnancy may also produce leucocytosis. In multiparæ it may be absent.

V. According to our observations, the leucocytosis post-partum averages about 18,000 on the day after delivery, gradually falling in the course of the next ten days. It is well to know that a considerable leucocytosis on the fifth or seventh day does not necessarily mean any abnormal amount of sepsis.

VI. The leucocytosis of the moribund state is moderate in degree, and is possibly due to stasis in many cases. In pernicious anæmia it may be extreme.

Pathological Leucocytosis.

For convenience' sake we divide the pathological leucocytoses into the following groups:

I. The leucocytosis after hemorrhage;
II. The leucocytosis of infection;
III. Intermediate between II. and IV.;
IV. Toxic leucocytosis;
V. The leucocytosis of malignant disease.

I. *After Hemorrhage.*—The degree of leucocytosis usually runs parallel with the degree of the anæmia, rather than with the amount of blood lost or the recency of the hemorrhage. Some cases will lose a quart of blood and be back to the normal number of red corpuscles

[1] *Zeit. f. klin. Med.*, 1895, p. 475. [2] *Loc. cit.*

within a day or two, especially after infusion of normal salt solution. Here the leucocyte count falls as the count of red cells rises. This leucocytosis has no diagnostic value, and is simply to be borne in mind to prevent the drawing of false inferences. Stengel finds a *lymphocytosis* after hemorrhage in some cases (see below).

II. The presence of the *leucocytosis of infection* seems to have very little relation to the *amount* or to the product of the inflammation where such is present. It is present, for example, in erysipelas, relapsing fever, cholera, follicular tonsillitis, malignant endocarditis, variola, and scarlet fever—diseases where the product is slight in amount—as well as in more productive processes, such as pneumonia, empyema, purulent meningitis and cerebro-spinal meningitis, appendicitis, and abscess of the liver. The small amount of pus contained in a felon may produce as great a leucocytosis as a general purulent peritonitis. Further, the *nature* of the inflammatory product seems to have but little influence on the degree of leucocytosis. The blood in diphtheria shows no greater increase of white cells than abscess of the tonsil, and neither differs much in this respect from gangrene of the lung or acute articular rheumatism. Counts in serous effusions into the pleural cavity range considerably lower than in purulent pleurisies, probably because most serous pleurisies are tubercular (see p. 693).

Other inflammatory conditions showing leucocytosis are—

Cholangitis, with or without gall-stones;
Empyema of the gall-bladder;
Abscess of the lung and advanced phthisis (with mixed infection) and abscesses of all kinds, such as perinephritic abscess, dorsal abscess, spinal or hip abscess, with mixed infections, etc.;
Acute nephritis, } in some cases;
Acute gastro-intestinal catarrh, }
Pericarditis—serous or purulent;
Osteomyelitis;
Pyæmia and septicæmia;
Dermatitis and all inflammatory skin diseases;
Endometritis (some cases);
Salpingitis and pelvic peritonitis;
Carbuncle.

III. Also in actinomycosis, glanders, acute yellow atrophy of the liver, rickets, and most anæmias of children, syphilis (congenital or in the secondary or tertiary stages), cirrhotic liver (some cases, especially in later stages), and acute peripheral neuritis.

The above group of conditions is intended to stand intermediate between the frankly inflammatory lesions before mentioned and those next to be enumerated, in which the presence of toxic or foreign substances in the blood seems to be the origin of the leucocytosis.

IV. *Toxic Leucocytosis*—(*a*) Diabetes (some cases);[1]
(*b*) Rickets (most cases);
(*b*) Uric acid diathesis;[2]
(*c*) Poisoning by illuminating gas;[3]

[1] Cited in *Klein, Volkmann's Sammlung klein. Vorträg.*, 1893, Dec.
[2] Neusser, *Wien. klin. Woch.*, Sept. 27, 1894.
[3] Eaton, *Boston Med. and Surg. Journal*, Mar. 18, 1895.

(*d*) After tuberculin injections and ergotin ;[1]
(*e*) After intravenous injections of normal saline solution ;[2]
(*f*) During and after etherization ;[3]
(*g*) After ingestion of salicylates.

Possibly the leucocytosis of acute delirium belong to this group, and perhaps one or more of group III. as well.

In animals the shock produced by a simple fracture or an aseptic laparotomy is sufficient to produce temporary leucocytosis. We are aware of no observations on this point in man.

V. *Malignant Disease.*—The leucocytosis of malignant disease very possibly belongs under one or another of the above headings, but in our present ignorance of the mode of production of *any* leucocytosis it is convenient to consider it separately.

As a rule, it is the disseminated and rapidly growing tumors that cause leucocytosis. In the more localized and slow-growing cancers, like those of the lip and breast and most of those of the stomach, there is no leucocytosis. Thirty out of 46 cases of gastric cancer observed at the Massachusetts General Hospital showed no leucocytosis. Those who do show it are usually the advanced and very cachectic cases with metastases. Cases with metastases in various organs and in the omentum usually show an increase of white cells.

Sarcoma follows about the same rules as cancer, but, as a general thing, the counts run higher. Small, slow-growing tumors without cachexia, no leucocytosis. Diffuse growths, sarcomatosis, and osteosarcoma give high counts.

The highest counts we have ourselves seen have been as follows :

Cancer of all abdominal organs (originating in stomach),						105,000
"	"	"	"	"	" kidney,	91,000
"	"	"	"	"	" pancreas,	89,000

Some cases of cancer of the œsophagus or the cardiac end of the stomach show a diminution in the number of leucocytes, owing to the starvation.

Of the absence of digestion-leucocytosis in gastric cancer we have already spoken.

Benign tumors, such as fibroma, lipoma, and myoma, usually produce no leucocytosis.

Absence of Leucocytosis.

Leucocytosis is conspicuously absent in—

Typhoid (uncomplicated),

Tuberculosis, { unmixed infections *except tubercular meningitis.* }, including { Incipient phthisis. / Tubercular peritonitis. / General miliary tuberculosis. / Hip disease. / Pott's " / " Cold abscess," etc. }

[1] Botkin, *Deut. med. Woch.*, 1892, No. 15.
[2] Löwitt, *Studien und Path. d. Blut, und Lymph.*, Jena, 1892.
[3] Klein, *loc. cit.*

Malaria.
Grippe (according to most observers).
Measles.
Leprosy.
Intestinal obstruction (non-malignant).
Also in most cases of—
Cystitis.
Endometritis.

In drawing conclusions for diagnostic or prognostic purposes for a leucocytosis we must first exclude the various physiological leucocytoses. Then it is almost as important to repeat the observations as it is in the use of the clinical thermometer. A single count or a single record of temperature tells but little. A temperature chart and a leucocyte chart tell a great deal.

For example, a steadily rising leucocyte chart in a case of *malignant disease* (Fig. 49) foretells the approach of the fatal termination. Similarly, the increase of the count of leucocytes in a case of appendicitis is of far more value than the simple knowledge that the count is high or low, indicating as it usually does that the inflammation is spreading. When the abscess has become thoroughly walled off, the count usually remains stationary or falls, and the subsidence of the trouble is likewise indicated by the fall in the leucocyte count.

When the drainage of any *abscess* is established, except tubercular abscesses, the leucocyte count falls somewhat, but seldom to normal, and any pocketing of pus later on is indicated at once by a rise in the count, often before it would have been discovered from other signs or symptoms.

This is illustrated by the accompanying chart (Fig. 50).

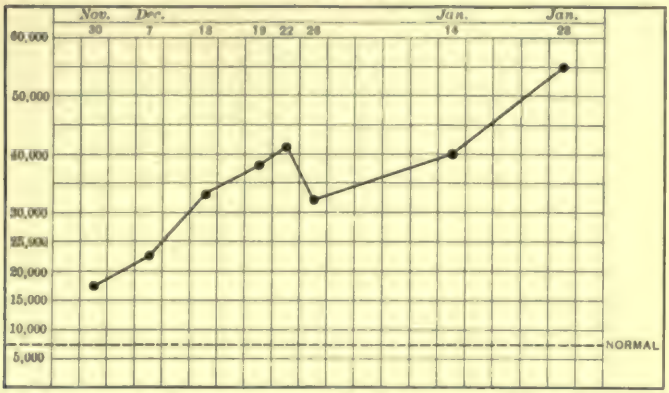
The leucocytosis of *pneumonia* is often of diagnostic importance in that it may appear before any physical signs of the disease can be detected, as in the so-called "central" pneumonias. In cases which would otherwise have seemed like grippe, bronchitis, or a feverish "cold" the presence of leucocytosis may make us suspect pneumonia, and the physical signs often appear later and confirm our suspicion. The absence of leucocytosis in any but the mildest cases is a very bad sign prognostically. Independent observations in all parts of the world confirm this. On the other hand, the presence of leucocytosis, large or small, is of no favorable import.

The leucocyte count begins to fall a little before the temperature in most cases ending by crisis, but reaches normal later than the temperature. In the so-called "pseudo-crises" the count does not fall—a point of importance in distinguishing the true from the false crisis. As a rule, the increase of white cells takes place in the polymorphonuclear cells at the expense of the lymphocytes and eosinophiles, but in a case of broncho-pneumonia occurring at the Massachusetts General Hospital in 1894 the differential count showed 66 per cent. of lymphocytes and only 30 per cent. of polymorphonuclear cells. The leucocytosis was 94,600 cells at its maximum, but fell with the temperature, and quick recovery followed.

In cases of non-resolution of the consolidated lung after the temperature has fallen, the leucocyte count remains high.

The absence of leucocytosis in *typhoid fever*, and its presence in

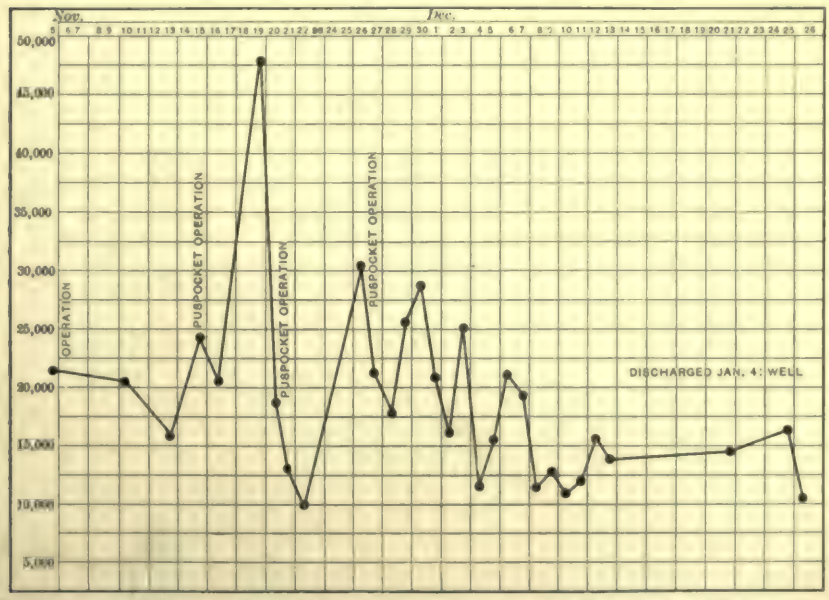
FIG. 49.



Showing the course of the leucocyte-count in a rapidly growing sarcoma of the kidney.

many of the affections with which this disease may be confounded, such as abscess of the liver, meningitis, central pneumonia, septicæmia, makes it of value in the diagnosis of this disease. On the other hand, as

FIG. 50.



Showing course of leucocyte count in a case of appendicitis, with pocketing of pus after operation.

between typhoid and grippe, tubercular peritonitis or general miliary tuberculosis, it gives no help. Here Widal's serum reaction is of great value.

Leucocytosis helps to tell whether a rise in temperature in the

course of a typhoid means relapse or some complication like otitis media, thrombosis, or furunculosis, the relapse not affecting the leucocyte count, while the others raise it.

Between *serous* and *purulent effusions* into the pleural cavity the blood count aids, in that serous effusions very rarely raise the leucocytes above 14,000, while empyema usually shows higher figures.

In a diagnosis between an abscess from *appendicitis* and a catarrhal appendicitis, intestinal obstruction, simple constipation, etc. there is some help in the fact that leucocytosis occurs in almost all cases with abscess, and not in the other various conditions simulating it.

In various obscure pelvic conditions in women with pain and fever, the presence or absence of leucocytosis often helps to decide whether pus is present or not. The same is true of cases where the diagnosis of osteomyelitic abscess is in doubt. In short, in any case where deep seated suppuration is a possibility the blood count cannot be neglected.

The absence of leucocytosis in *measles* and its presence in *scarlet fever* are often of help in doubtful cases. In scarlet fever the leucocytosis may precede the rash by several days.

Between *malignant disease* and *tuberculosis* the blood sometimes aids, but oftener does not, for neither condition causes leucocytosis in early cases, where doubt usually arises; and if there is a leucocytosis, we cannot rule out tuberculosis, for the infection may be a mixed one, and so raise the leucocyte count as much as malignant disease would do. Spinal and hip abscesses show low counts until they are opened and the injection mixed. Afterward the count rises.

Between malignant disease and pernicious anæmia the blood examination may be the only means of deciding. In a recent case with every *symptom* of pernicious anæmia and no localizing symptom or sign, our diagnosis of malignant disease rested wholly on the presence of marked leucocytosis without any great diminution in the red cells, and was confirmed by autopsy. The extreme pallor here was due to lack of hæmoglobin. There was no pain or emaciation.

It remains to be said that in some cases of *purulent peritonitis* and other *severe septic conditions* leucocytosis is sometimes entirely absent. These cases, like those of pneumonia with normal blood, are always fatal, so far as we know, and it is interesting to note in this connection that it has been found by several observers that animals who succumb at once to a heavy dose of a culture of pathogenic cocci do not show the leucocytosis which is the rule in those who survive.

It seems as if the cases in which the system and the infection are well balanced and the struggle between them hard, have leucocytosis, while an easy victory for either side (very mild or overwhelming infections) prevents the count from rising.

HODGKIN'S DISEASE.

DEFINITION.—A disease characterized by a progressive hyperplasia of the spleen, lymphatic glands, or both, with or without secondary lymphatic growths in various parts of the body.

Synonyms.—Pseudo-leucæmia; Malignant lymphoma; Splenic anæmia; Lympho-sarcoma.

Etiology.—The etiology is unknown. It is so difficult to distinguish certain cases of the disease from glandular enlargements due to tuberculosis or syphilis that some writers have supposed all cases to be of this nature, but the evidence against this view is strong. It is commoner in males and in persons under forty years of age, and seems to be endemic in certain localities in the Tyrol. An infective cause is suggested by certain features of the disease, but the evidence is insufficient. Some cases are said to develop into leucæmia.

Pathological Anatomy.—The lesions are in the main identical in character with those of leucæmia (see p. 687), although in the majority of cases Hodgkin's disease begins in the glands, especially those of the neck, and affects the spleen slightly if at all. Nevertheless, there occur cases of Hodgkin's disease where the spleen is practically the only organ affected and grows to the same dimensions as in the worst cases of leucæmia, and slight changes in this viscus are present in three fourths of the cases.

The bunches and masses of glands formed especially in and near the neck and mediastinum are apt to be larger than are met with in leucæmia.

The glands are softer in the early stages of their growth, when cellular elements are relatively abundant and connective tissue is scarce. Later, as the latter increases in amount, the glands get harder and become matted together.

The disease usually remains limited within the capsule of the gland, but sometimes the capsule is perforated and periglandular proliferation ensues.

A greenish color may be seen in the tumors, as in those of leucæmia—an appearance to which the rather superfluous name of chloroma has been given.

The tumors in the thymus, thyroid, lungs, liver, kidney, gastro-intestinal tract, and skin may occur as in leucæmia, and do not differ from the latter in any respect.

As to the changes in the bone marrow, there is nothing to add to the account given under Leucæmia (p. 688).

Symptoms.—Practically, the symptomatology amounts to—

(*a*) Pressure symptoms;

(*b*) Symptoms of anæmia.

(*a*) The *pressure symptoms* are usually the more prominent. They may be as various as the number of situations in which lymphatic tissue exists. In the majority of cases, however, as they are seen clinically, the cervical glands are most affected, and it is for them that the patient seeks advice.

They are not painful at first, and it is the disfigurement and inconvenience resulting from them, as well as a certain amount of debility, of which the patient complains. He does not usually take much notice of the inguinal and axillary glands, which are generally affected to a lesser extent than the cervical. In the rare cases with acute febrile course the general symptoms may be very marked. Pain, headache, and congestion of the face may result sooner or later from the progressive enlargement of these glands, or they may remain for an indefinite

period of time without enlargement and without appearing in any other part of the body.

Where the glandular enlargement invades the throat we may have dyspnœa or dysphagia, and similar symptoms may occur from the pressure of bronchial or mediastinal glands. Deafness may result, as in leucæmia, from growths in the internal ear or naso-pharynx. More common is a certain amount of unilateral flushing, sweating, or inequality of the pupils due to pressure on the cervical sympathetic. Stridor or aphonia from pressure on the recurrent laryngeal is sometimes seen. Where the axillary or inguinal glands are very large the patient may complain of swelling of an extremity; pressure on nerves may cause severe and constant pain. This last symptom is, however, on the whole, surprisingly infrequent.

Ascites from the growths in the mesenteric glands or those about the hilus of the liver has been observed.

This catalogue of symptoms might be much extended.

(*b*) *The anæmia* may be very moderate or even absent over long periods, but if the pressure symptoms do not lead to a rapid downward course, the symptoms already described under the anæmias usually appear. Lymphoid tumors in the mouth or intestine may give rise to ulceration. Fever is about as constant and of the same intensity as in leucæmia. Periods of irregular fever may alternate with apyretic intervals. We have observed occasionally the same unexplained chills as in leucæmia and pernicious anæmia.

Physical examination is concerned chiefly with the various sets of enlarged glands and the negative condition of the blood. Large irregular tumors in the neck are common, usually not tender or only in a few small areas. The growths are generally much larger on one side of the neck than on the other. A number of irregular nodules can be made out, matted together and usually closely adherent to the skin. The superficial veins may be dilated in compensation for the cutting off of the circulation through deeper vessels. Smaller kernels of similar nature are to be felt in the axilla and groins, as a rule, and sometimes large masses can be detected through the abdominal wall. Where a primary bronchus is pressed on we may have stridor or complete absence of respiration in one lung, and dulness and absence of breathing in the upper front part of the chest are often caused by masses of enlarged mediastinal and bronchial glands.

Lymphoid nodules may appear in the skin—elevated irregular reddish lesions which may ulcerate. Hemorrhages under the skin may be present, especially in the acute forms, in which they may be so numerous as to resemble Werlhof's disease.

The heart shows nothing, unless the murmurs common to all anæmic conditions.

The spleen may be very considerably enlarged, with or without glandular hypertrophy. In a case recently seen by us it reached nearly to the navel, and lesser degrees of enlargement are common. Its other characteristics are those of leucæmic spleens. According to the classification here adopted, all cases of enlarged spleen without known cause are considered Hodgkin's disease, except where all signs and symptoms of debility are absent, when the term "simple hypertrophy" is used.

"Splenic anæmia" is either a form of Hodgkin's disease or an anæmia secondary to some such cause as malaria, rickets, or syphilis, and accompanied by splenic enlargement. The liver is also not infrequently enlarged.

As in leucæmia, bronzing of the skin may occur—possibly, as has been suggested, from pressure on the adrenals or from continued use of arsenic.

The Blood.—The absence of any increase of the white cells is the important feature. Toward the end of the disease or in case of any suppuration in the enlarged glands we expect a slight leucocytosis, the increase taking place solely in the polymorphonuclear cells.

The red corpuscles are usually not much diminished. Occasionally at the end of the disease they fall as low as 2,000,000, the hæmoglobin being proportionally affected.

The stained specimens sometimes show an increase of lymphocytes, and in one case, affecting chiefly the spleen, we saw a large increase of the large mononuclear forms.

Myelocytes are sometimes present in small percentages, as they are in any cachectic disease. Occasionally a few nucleated red cells (normoblasts) are seen.

DIAGNOSIS.—The disease can be identified only by excluding such other diseases as are accompanied by enlargement of the glands or spleen. This is sometimes impossible until autopsy, and then only by the most careful inoculation experiments. The appearances and symptoms of the enlarged glands may be identical with those of tubercular adenitis, even post-mortem, and bacilli may be so scanty that only the inoculation of animals with pieces of gland substance will demonstrate their presence.

Softening is common in tuberculosis and rare in Hodgkin's disease, but exceptions occur. The discovery of tuberculosis elsewhere is of assistance, but is not conclusive evidence against Hodgkin's disease, since the two may coexist.

Primary new growths of the glands (lympho-sarcoma) are much more apt to be accompanied by marked leucocytosis, but no satisfactory distinction has been established between the two diseases, either clinically or by the microscope.

Syphilitic adenitis can usually be excluded by the history and by the other manifestations of syphilis.

Splenic Hodgkin's disease is recognized when malaria, leucæmia, rickets, and amyloid disease can be excluded as causes of the cachexia and splenic tumor. Simple hypertrophy of the spleen without any other symptoms or signs may occur as above said, and does not merit the title of Hodgkin's disease. The cause of the hypertrophy in such cases is unknown.

From leucæmia the disease is distinguished solely by the blood examination.

PROGNOSIS AND COURSE.—As above mentioned, there are cases which remain confined for many years to one set or mass of glands and give no considerable inconvenience to the patient. The glands show scarcely any tendency to suppurate. Another set of cases shows a slow but steady increase in the size of the masses, without any bad pressure

symptoms, and these cases may hang on for five or six years, when anæmia, exhaustion, or the advent of pressure symptoms leads to death. The appearance of intrathoracic growths makes the outlook grave. Still another group of patients suffer from a truly acute form of the disease, which has been studied especially by Westphal and Ebstein. A case of this kind lately came under our notice where the whole course of the disease occupied only seven weeks.

TREATMENT.—Arsenic again is our chief instrument against the disease. Under its use the glands sometimes seem to grow smaller and the other symptoms to improve proportionally. It may be given in the form of Fowler's solution, 2 minims after meals, increasing the doses up to the limit of toleration. Arsenic has also been used hypodermically, a few minims of Fowler's solution being injected directly into the gland masses, which causes pain and sometimes some inflammatory reaction. When this subsides the injection may be repeated.

Favorable results are reported from the combined hypodermic and internal use of the drug. The question of removing the glands by operation is sometimes to be considered. Where the disease seems to be localized in one or two places it is well to remove the glands in order if possible to diminish the likelihood that the disease will spread. Appearances may, at any rate, be improved and pressure symptoms abated. Where there is evidence that the mediastinum or internal organs have been involved operation is not to be advised. In general the treatment is that of debility, the attempt being to place the patient at the height of his powers of resisting the disease by hygienic and supportive treatment. Iron is not indicated unless there is actual anæmia as shown by blood examination.

THE BLOOD IN INFANCY.

DURING the earliest days of life both red and white corpuscles are considerably above the normal of adult life, 6,000,000 to 8,000,000 being not unusual for the red cells and 20,000 to 30,000 for the white. The hæmoglobin is also greatly increased, and often is too high to be measured at all by v. Fleischl's instrument. The following table shows the difference in the percentage of the several varieties of leucocytes in adults and young infants:

	Adults.	Infants.
Polymorphonuclear neutrophiles	60–75 per cent.	25–40 per cent.
Lymphocytes	18–30 "	40–60 "
Large mononuclear and transitional	4–8 "	6–12 "
Eosinophiles	½–4 "	1–10 "

These differences in the count and in the proportions of different varieties of leucocytes are important to bear in mind in any question of leucocytosis in infancy. Thus a count of 20,000 white cells is no leucocytosis at all in the first two weeks of life, and 70 per cent. of polymorphonuclear cells is a marked increase, though it would be normal in

adults. Scanty normoblasts are occasionally to be found in healthy infant's blood during the first days of infancy.[1]

All these modifications gradually disappear at a rate varying with the rapidity of the child's development, but, as a general rule, the red cells sink to normal within a few days, while the white continue more or less above normal until about the sixth year.[2] As the whole number of leucocytes diminishes, the percentage of the young cells (lymphocytes) diminishes proportionally and the polymorphonuclear cells rise. The high percentage of lymphocytes in various children's diseases has sometimes been known as "*lymphocytosis*," but it is better thought of as retrograde metamorphosis. Such changes are observed on the outbreak of hereditary syphilis, rickets, scurvy of infancy, and in some of those conditions known as infantile atrophy.

Anæmia in Infancy.

1. It seems to need less to start an anæmia in infancy than in adult life.[3] The serum is at that age especially toxic, and the red cells perhaps abnormally vulnerable. Slight diarrhœal or catarrhal affections are sometimes followed by profound and even fatal anæmia. It may be that adults exemplify by the comparative resistance of their blood to drains upon the general vitality the survival of the toughest. Perhaps those whose blood-making organs are comparatively unresponsive to demands for new blood stock never reach adult life.

2. Another characteristic of infantile anæmia is the tendency to be accompanied by enlargement of the spleen. Hence the term "splenic anæmia," which refers mostly to cases occurring in infants and young children.[4] It does not appear to represent any separate type of anæmia, for it is a characteristic of all the anæmias (and indeed of all blood diseases) of infancy. Thus it is present in many of the anæmias associated with rickets and hereditary syphilis, and commoner in the pernicious anæmia of infants than in adults.

3. A third characteristic of infantile anæmia is the common occurrence of leucocytosis—often to be explained, as we believe, as a reversion to the blood typical of an earlier age.

4. Myelocytes appear to be more common in the anæmias of infancy than of adults, and are not confined to pernicious cases, but appear in the various secondary anæmias.

Anæmia Infantum Pseudo-leucæmica.

It is partly owing to these peculiarities that a separate and peculiar anæmia of infancy has been described by v. Jaksch,[5] Luzet,[6] and other writers—the so-called "anæmia infantum pseudo-leucæmica." In our opinion the cases so described belong either under the head of pernicious anæmia with leucocytosis or of leucæmia. Myelocytes are present in

[1] Fischl, *Zeitschrift f. Heilk.*, 1892. [2] See table in Rotch's *Pædiatrics*, p. 342.

[3] (*a*) Weiss, *Jahrb. f. Kinderheilk.*, 1893, vol. xxxviii.; (*b*) Monti and Berggrün, *Die Chron. Anæm. d. Kindesalter.*, Leipzig, 1892.

[4] Cases of the splenic form of Hodgkin's disease in adults have also gone under this title (p. 699).

[5] *Prag. med. Woch.*, 1890. [6] *Dissert.*, Paris, 1891.

considerable numbers in both diseases, and in both the total count of leucocytes is or may be greatly increased. Splenic enlargement is common to both, so that it must be acknowledged that there are cases in infancy which it is very hard to name—leucæmia or pernicious anæmia. In fact, it is here that we have the strongest evidence for the belief that pernicious anæmia and leucæmia are different types or branches of one disease, the intermediate stages appearing chiefly in infancy.

The "polymorphous" character of the blood referred to by v. Jaksch in his description of the disease is not greater than appears in certain adult cases of pernicious anæmia or leucæmia, and is due to the presence of many varieties of nucleated red corpuscles, together with the normal varieties of white cells and the degenerative forms of both kinds. It is impossible in some cases to distinguish with Ehrlich's stain between certain forms of megaloblasts and certain atypical lymphocytes, and the general impression is that every cell in the field is very different from every other. Nevertheless, as above said, there are no cells present that are not to be found in the adult cases of leucæmia or pernicious anæmia.

DISEASES OF THE KIDNEYS.

DISEASES OF THE KIDNEYS.

NEPHRITIS; AMYLOID DEGENERATION OF THE KIDNEYS; RENAL HYPERÆMIA.

By HENRY P. LOOMIS, M. D.

NEPHRITIS (BRIGHT'S DISEASE OF THE KIDNEYS).

Definition.—Bright's disease of the kidneys is a clinical name, implying a disease in which the urine exhibits certain morbid changes. The name was given originally as an appropriate acknowledgment to Dr. Richard Bright for his discovery in 1827 that certain cases of dropsy associated with albuminuria were due to disease of the kidneys, and has been understood by some to include all renal affections associated with albuminuria; by others, simple degenerations and disorders of renal circulation.

The writer believes that, in the light of our present pathological knowledge, the term Bright's disease should be discarded, and the name nephritis substituted, and under this name should be included all inflammations of the kidney non-suppurative in character. The adjective "diffuse" prefixed to the word "nephritis" shows at once that the inflammatory changes involve both kidneys, and so is not a suppurative inflammation which would be circumscribed. It also implies that all the anatomical elements of the kidney are involved in the inflammatory process, which is now known to be the fact in all forms of Bright's disease. The writer will therefore use the term diffuse nephritis in place of the generally accepted term Bright's disease, and would note the following facts:

1. That in all cases of diffuse nephritis both kidneys are affected, and generally to about the same extent.

2. Different portions of the same kidney are not changed to the same extent. Even in advanced nephritis normal areas sometimes remain.

3. The exciting cause of all forms of diffuse nephritis reaches the kidneys by the bloodvessels and produces first inflammatory alterations in the vessel walls. The cause being hæmatogenous, therefore, all elements are more or less involved. Bright's disease may thus be defined as a bilateral hæmatogenous non-suppurative nephritis.

Not every case of albuminuria, even with a few hyaline casts, is to be regarded as a true nephritis.

The writer has been especially impressed with the fact that a very large proportion of the patients who give no renal symptoms yet have albumin and casts in the urine. When it becomes the routine practice to examine the urine of every patient under observation, physicians will be surprised to find how often albumin and casts appear in the urine when no kidney changes are suspected. A medical examiner of one of the largest insurance companies found albumin in the urine of 11 per cent. of the persons applying for insurance, this being the only evidence of any abnormal condition.

Albuminuria appearing at intervals, while it does not indicate that a nephritis has developed, shows a certain functional weakness of the kidneys, especially of the glomeruli, which should always cause some anxiety; and especially is this so if hyaline casts are simultaneously found. These latter indicate one step farther on the road which may lead to diffuse nephritis.

It is the exception to find kidneys in persons over forty years of age which will show *under the microscope* even a relatively normal structure. In the lower walks of life nearly all kidneys are diseased. This may seem to approach a broad statement of fact, but it is based on an experience of over ten years as Curator of the City Morgue and a personal examination of over two thousand cases. An examination of a large number of coroner's cases, many of which are sudden deaths while in apparent perfect health, also bears this out. In my last 250 autopsies were only 10 cases in which the kidneys were *perfectly* normal.

Etiology plays a secondary part in the development of nephritis. This depends upon the fact that every nephritis is toxic and depends upon changes in the blood. This may be due to (1) the toxins of micro-organisms, as in diphtheria; (2) pathological products of metabolism, as intestinal ptomaïnes; (3) specific kidney poisons, as cantharides; (4) products of tissue waste, as uric acid.

The results, the anatomical types, and the clinical pictures of the disease depend upon the quantity, quality, and duration of the acting poison.

Classification.—It is essential in the study of nephritis that some classification should be adopted. Writers differ widely as to the anatomy and pathogenesis of nephritis. It seems inadvisable to discuss the various classifications that have been proposed. Some are based on the clinical aspect of the disease, some are considered from the etiological standpoint, and others from the gross appearance of the kidneys. All of these are uncertain and are open to personal interpretations. For practical purposes the writer has adopted the following classification as being the most reliable and one approved by clinical experience. It is based on the *exact changes* which have taken place in the kidneys as revealed by the microscope. It is only by the microscope that we can positively tell all the changes which have taken place in the renal tissue as the result of this disease.

All nephrites can be divided into two great divisions—acute diffuse nephritis and chronic diffuse nephritis, the former generally ending in

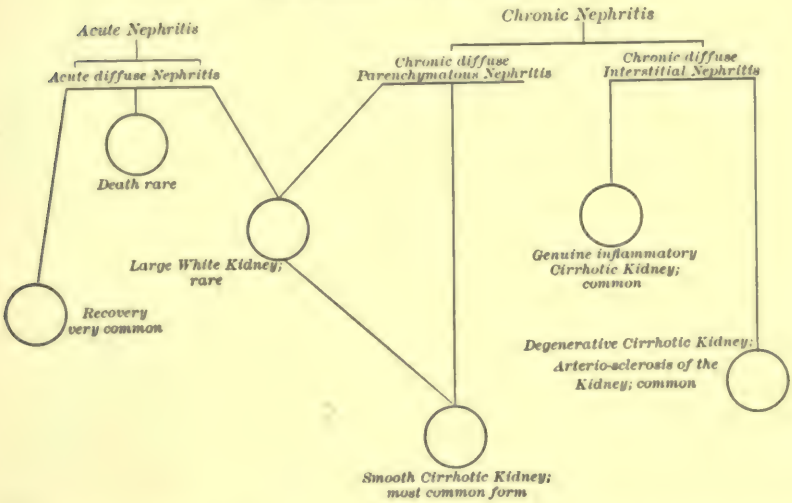
recovery, the latter always in death—the former being comparatively rare, the latter very frequent.

Chronic diffuse nephritis is again subdivided into—(1) chronic diffuse parenchymatous nephritis, in which the tubules and epithelial elements are primarily and permanently affected—a form which follows the acute or comes on insidiously; (2) chronic diffuse interstitial nephritis, in which the inflammatory changes affect principally the connective tissue of the organ.

The advantage of the above classification is that the clinical picture of each form of kidney is generally distinct. With an accurate history

FIG. 51.

Classification of Nephritis



of any case, especially as to the beginning of the trouble, the observer will have but little difficulty in foretelling exactly what kind of kidney will be found at autopsy. In the writer's opinion it is far ahead of any other classification, appealing at once to the clinician and the pathologist. It must be remembered that, after all, some cases of chronic nephritis cannot at once fall within the classification. Transitional periods must be considered, for "natura non facit saltus."

It has been argued that arterio-sclerosis is not a form of nephritis, but at the bedside its picture is clearly that of a nephritis.

It is a question with some observers whether the forms of nephritis are only different stages of the same process, or whether they are from the start essentially different, one having no relation to the other. Upon a clinical basis alone it is not difficult to decide the question, and pathologically it can be proven that the different forms of kidney seen at the autopsy start as distinct processes. Most cases begin as a chronic inflammation without at any time presenting an acute stage. The true cirrhotic kidney is always primary, and never comes from the acute or chronic parenchymatous form.

The following tables represent the results of the gross and microscopical examination of kidneys removed from 54 cases; the examination was undertaken by the writer with the following objects:

(1) To determine the relation between the gross and microscopical appearance of the kidneys.

(2) To determine the condition of the heart associated with the different varieties of kidney lesions.

(3) To determine the relation between the chemical and microscopical results of the examination of the urine during life, and the microscopical lesions of the kidney found after death.

The kidneys were grouped according to the classification just given, the diagnosis being made from a careful examination of the microscopical sections taken from different portions of the kidneys.

Number of cases examined, 54; normal kidneys, 8; acute diffuse nephritis, 12; chronic diffuse parenchymatous nephritis, 22; chronic diffuse interstitial nephritis, 12.

Gross Appearance of the Kidneys.—The following is the gross appearance of the kidneys. Each kidney has been classified according to the results of the microscopical examination:

Normal Kidneys, 8.

Enlarged, 4.

Sex.	Age.	Wt. oz.	Color.	Capsule.	Cortex.	Markings.	Urine.	Remarks.
Female.	20	5½	Normal.	Not adherent.	Normal.	Indistinct.	Sp. gr. 1021. Albumin and casts.	Phthisis, cause of death.
Male.	48	7	Red.	Not adherent.	Increased.	Indistinct.	Clear, sp. gr. 1014. No albumin or casts.	Phthisis, cause of death.
Male.	1	11	Normal.	Not adherent.	Increased.	Distinct.	. . .	Dropped dead in street.
Male.	40	7	Normal.	Not adherent.	Normal.	Distinct.	. . .	Acute dilatation of right heart. Found dead in street.

Normal Size, 4.

Male.	55	5	Red.	Not adherent.	Normal.	Indistinct.		
Female.	70	4½	Pale.	Slightly adherent.	Diminished.	Fairly distinct.	. . .	Died of senility.
Male.	28	4½	Pale.	Not adherent.	Normal.	Distinct.	No casts or albumin.	Suicide.
Male.	20	5	Normal.	Not adherent.	Normal.	Indistinct.	. . .	Arsenic-poisoning.

Acute Diffuse Nephritis, 12.

Enlarged, 6.

Sex.	Age.	Wt. oz.	Color.	Capsule.	Cortex.	Markings.	Remarks.
Female.	37	6	Red.	Not adherent.	Increased.	Indistinct.	Carcinoma of liver.
Male.	27	8	Pale.	Not adherent.	Diminished.	Obliterated.	Tubercular pneumonia.
Male.	40	7	Pale.	Not adherent.	Diminished.	Indistinct.	Pneumonia.
Female.	40	7	Dark.	Not adherent.	Increased.	Distinct.	Softening in right motor tract of brain.
Male.	59	6	Dark.	Not adherent.	Normal.	Distinct.	Uræmic dyspnœa and dilated heart.
Female.	30	8	Pale.	Not adherent.	Increased.	Indistinct.	Alcoholism and fatty heart.

Normal Size, 6.

Sex.	Age.	Wt. oz.	Color.	Capsule.	Cortex.	Markings.	Remarks.
. .	. .	. .	Pale.	Not adherent.	Diminished.	Indistinct.	Rupture, aneurysm.
Female.	67	4	Pale.	Not adherent.	Diminished.	Indistinct.	Phthisis.
Male.	18	4½	Pale.	Not adherent.	Normal.	Normal.	Cardiac hypertrophy, rheumatic history eight years.
Female.	30	4	Pale.	Not adherent.	Increased.	Indistinct.	
Male.	70	4½	Pale.	Not adherent.	Diminished.	Obliterated.	Tuberculous pneumonia.
Female.	20	5½	Red.	Not adherent.	Normal.	Indistinct.	Phthisis.

Chronic Parenchymatous Nephritis, 22.

Enlarged, 11.

Sex.	Age.	Wt. oz.	Color.	Capsule.	Cortex.	Markings.	Urine.	Remarks.
. .	. .	9	Red.	Not adherent.	Increased.	Obliterated.	Sp.gr.1029; gran. casts. No albumin.	
Male.	30	7	Dark.	Not adherent.	Normal.	Indistinct.	. . .	Interstitial myocarditis.
Male.	28	6½	Pale.	Not adherent.	Increased.	Indistinct.	. . .	"Large white kidney."
Male.	48	6	Pale.	Not adherent.	Diminished.	Obliterated.	Few granular casts.	
Male.	60	6	Dark.	Not adherent.	Thin.	Fairly distinct.	. . .	Chronic tuberculosis.
Male.	65	8	Dark.	Not adherent.	Increased.	Distinct.	No casts or albumin.	Cirrhosis of liver; ascites.
Male.	40	8	Pale.	Not adherent.	Increased.	Indistinct.	. . .	"Large white kidney;" tricuspid regurgitation.
Male.	35	7	Dark.	Not adherent.	Increased.	Distinct.	. . .	Grippe; pneumonia.
Male.	63	6	Normal.	Not adherent.	Normal.	Indistinct.	. . .	Tuberculous pneumonia; delirium tremens.
Male.	26	6½	Pale.	Not adherent.	Increased.	Indistinct.	Sp. gr. 1024. No albumin or casts.	Jaundice.
Female.	35	. .	Pale.	Slightly adherent.	Normal.	Indistinct.	. . .	Puerperal sepsis.

Normal Size, 8.

Male.	38	4½	Pale.	Not adherent.	Diminished.	Obliterated.		
Female.	50	4½	Dark.	Not adherent.	Normal.	Distinct.	. . .	Erysipelas; chronic alcoholism.
Female.	37	4	Pale.	Slightly adherent.	Increased.	Distinct.	. . .	Cardiac disease.
Female.	90	4	Pale.	Slightly adherent.	Increased.	Fairly distinct.	. . .	Senility.
Female.	80	4	Pale.	Not adherent.	Diminished.	Indistinct.	. . .	Senile; pneumonia.
Female.	20	4	Pale.	Not adherent.	Increased.	Indistinct.	. . .	Acute tuberculosis.
Male.	78	5	Pale.	Adherent	Diminished.	Obliterated.	Sp.gr.1018; gran. casts. Much albumin.	Tuberculosis.
Male.	63	4½	Pale.	Not adherent.	Normal.	Distinct.		

Small, 2.

Male.	45	3	Dark.	Not adherent.	Diminished.	Indistinct.	. . .	Œdema of legs; heart 31 oz.
Female.	64	3	Normal.	Not adherent.	Diminished.	Indistinct.	Sp. gr. 1018. Albumin.	
Female.	28	3½	Dark.	Not adherent.	Diminished.	Indistinct.	Albumin. Sp.gr. 1028; casts.	Ill 10 weeks; anæmic, uræmic.

Interstitial Nephritis, 12.

Enlarged, 8.

Sex.	Age.	Wt. oz.	Color.	Capsule.	Cortex.	Markings.	Urine.	Remarks.
Male.	55	8	Red.	Not adherent.	Diminished.	Fairly distinct.		
Male.	75	6	Pale.	Not adherent.	Diminished.	Indistinct.	. . .	Large aneurysm.
Male.	48	9	Pale.	Adherent	Increased.	Indistinct.	. . .	Heart 30 oz.; cardiac disease.
Male.	60	5	Pale.	Not adherent.	Increased.	Indistinct.		
Male.	66	7	Pale.	Slightly adherent.	Increased.	Obliterated.	Sp. gr. 1018. Much albumin.	Phthisis.
Male.	34	8	Dark.	Not adherent.	Increased.	Indistinct.	. . .	Uræmia.
Male.	40	7	Pale.	Not adherent.	Increased.	Indistinct.	Casts, hyaline and gran.; albumin.	Pneumonia.
Male.	39	6	Pale.	Not adherent.	Increased.	Fairly distinct.	. . .	Lobar pneumonia.

Normal Size, 3.

Female.	47	4½	Pale.	Slightly adherent.	Diminished.	Obliterated.	. . .	Alcoholism and uræmia.
Male.	40	5	Pale.	Not adherent.	Diminished.	Indistinct.	Sp. gr. 1018; gran. casts. Much albumin.	Interstitial myocarditis.
Female.	70	4	Pale.	Adherent	Diminished.	Indistinct.		

Small, 1.

Female.	84	3	Pale.	Adherent	Diminished.	Indistinct.	. . .	Tubercular pneumonia; senility.

Condition of the Heart.—In 30 cases a careful examination of the heart showed the following abnormal condition:

(1) Heart cavities (especially left ventricle) dilated, with no valvular lesion in 8 cases; 3 of these cases were acute diffuse nephritis; 3 cases were chronic diffuse parenchymatous nephritis; 2 cases were chronic diffuse interstitial nephritis.

(2) Heart cavities dilated, with some thickening of the valves, in 22 cases. In some cases the changes were slight, and in all the heart lesion was apparently not primary. Three of the cases were associated with acute diffuse nephritis; 12, chronic diffuse parenchymatous nephritis; 7, chronic diffuse interstitial nephritis.

The following is the condition of the heart observed in 47 of the cases:

Condition of the heart.	Condition of the kidneys.				
	Normal.	Acute diffuse nephritis.	Chronic parenchymatous nephritis.	Chronic diffuse interstitial nephritis.	Total.
Good	5	5	7	2	19
Bad	2	7	9	10	28
	7	12	16	12	47

As the terms *good* and *bad* can only be relative terms, under the former are embraced normal hearts or with so slight changes noted that there could have been no cardiac symptoms observed during life. Under the term *bad* are embraced hearts with more or less changes about the

valves, in the muscular tissue of the heart, and in the size of the cavities—hearts in which the cardiac symptoms were permanent during life.

Condition of the Urine.—In 15 cases repeated examinations of the urine were made during life, and a classification of the kidneys removed at autopsy from these cases was made, based on their microscopical examination. The following is the result:

Condition of the urine.	Classification of the kidneys.				
	Normal.	Acute diffuse nephritis.	Chronic diffuse parenchymatous nephritis.	Chronic diffuse interstitial nephritis.	Total.
No albumin or casts	2	0	2	0	4
Albumin and casts (hyaline and granular)	1	3	4	3	11
	3	3	6	3	15

In 1 of these cases casts were found in the tubules of the kidney.

Besides the 15 cases just enumerated are the following 6 cases, which were under observation for long periods of time before death, and whose urine was repeatedly examined. The average results of the urinary examinations are here given, as also the gross and microscopical appearances of the kidneys removed from these cases after death:

Urine Analysis.

Case.	Reaction.	Color.	Albumin.	Casts.	Other products.
1	Acid.	Amber, cloudy.	25% (by vol.).	Hyaline and granular.	Leucocytes, epithelial cells.
2	1035 sp. gr.; acid.	Light yellow.	None.	Epithelial.	Large amount sugar.
3	1010 sp. gr.; neutral.	Reddish yellow.	.7–.8% (vol.).	Hyaline.	Pus cells, granular matter.
4	1020 sp. gr.; clear.	. . .	Small amount.	Hyaline and gran.	None.
5	1029 sp. gr.; acid.	Dark.	None.	Granular.	Crystals, uric acid.
6	1017 sp. gr.; acid.	Cloudy yellow.	.7% albumin.	Hyaline and gran.	None.

Gross Appearances, Kidney.

Case.	Capsule.	Cortex.	Color.	Weight.	Markings.
1	Non-adherent.	Thick.	Pale.	5 oz.	(Depressed cicatrices), coarse and granular.
2	Adherent.	Diminished.	Red.	Small.	(Calyces filled with fat), obliterated.
3	Non-adherent.	Normal.	Red.	4½ oz.	Obliterated.
4	Congested.	"	Red.	Small.	Indistinct.
5	Non-adherent.	Thick.	Red.	9 oz.	Obliterated.
6	Adherent.	Diminished.	Yellow.	6 oz.	"

Kidney (Microscopical Diagnosis).

Case.	Lesion.	Connective tissue.	Bloodvessels.	Tubular epithelium.
1	Early cirrhosis.	Slight increase.	Intra-capillary glomerulitis.	Slight granulo-fatty metamorphosis.
2	Diffuse cirrhosis.	Everywhere bet. tubules.	Endarteritis obliterans.	Cloudy swelling.
3	Cirrhosis.	Normal.	Normal.	Hyaline casts in tubes; great degeneration.
4	Chron. parenchymatous.	No increase.	Normal.	Free borders of cells gone.
5	Chron. parenchymatous.	No increase.	Capillaries congested.	Tubes more or less denuded.
6	Chron. parenchymatous.	Some slight increase.	Endarteritis obliterans; amyloid (?) in arteries.	Tubes dilated; hyaline casts, granular matter.

Acute Diffuse Nephritis (Acute Bright's Disease).

Definition.—Acute diffuse nephritis is an acute inflammation of the kidneys, called *diffuse* because all the anatomical elements of the organ may be involved, although practically it is generally the epithelial cells which are affected. This form of nephritis has been called by various writers catarrhal, parenchymatous, desquamative, croupous nephritis, glomerulitis, acute tubal or exudative nephritis.

The acute is a much rarer form of the disease than the chronic. Examinations under the microscope of 54 kidneys removed from suspected cases of nephritis, made by the writer during six months of 1896, showed only 12 examples of acute nephritis. Many cases diagnosed from the clinical symptoms as acute Bright's disease showed upon microscopical examination that they were acute attacks engrafted upon chronic forms of the disease. While the clinical picture does not vary to any great extent whether the disease occurs primarily or secondarily, still the prognosis depends in many cases on the ability to decide this question, and I know of none at times more difficult to determine. Often pseudo-primary forms occur where the disease, such as scarlet fever, which started the kidney changes was so mild as to be overlooked.

Anatomical Characters.—**Gross Appearance.**—The kidneys of acute nephritis may be divided into two classes according to their color: (1) *the pale kidney;* (2) *the dark red kidney.* They are either of normal size or enlarged, never small. Their capsule is always non-adherent and their surface smooth—a fact which is of aid in diagnosis.

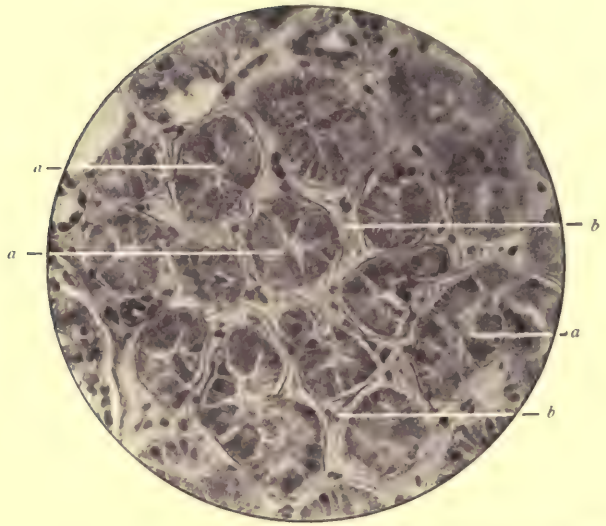
The red kidneys are firm, the pale ones may be soft. The variations in color of the kidneys do not represent different varieties of the disease, but are due to the amount of blood in the organs and the extent of the changes in the tubes at the time of death. The pale kidney represents, ordinarily, a later stage of the disease. The writer has examined with the microscope kidneys whose appearance to the naked eye was perfectly normal, and found well marked changes of acute nephritis. Such kidneys, however, are rare. Pale kidneys are relatively or absolutely anæmic, and often are of a yellowish color. Small hemorrhages or congested stars of Ferrein are seen on their surface. On the cut surface the darker medullary portion is in marked contrast to the pale cortex. Generally the glomeruli are congested and can be distinctly seen. The cortex is swollen and the normal markings are obliterated.

Dark Red Kidney.—These are the most common kidneys seen in acute nephritis, the color depending upon the intense hyperæmia. Through these kidneys are scattered hemorrhagic portions where the red color is intense. The medullary portion is always more intensely red than the cortex. These kidneys are softer than normal, moist, and the distinct markings are generally obliterated. The glomeruli stand out prominently as minute red points.

Microscopical Appearance.—1. *Mild Cases, not Fatal.*—In these cases the changes are often astonishingly slight, although the urine *before death* may have been loaded with albumin, casts, and blood. This is especially apt to be seen in scarlatinal nephritis, where the changes are confined principally to the glomeruli. In a series of 15

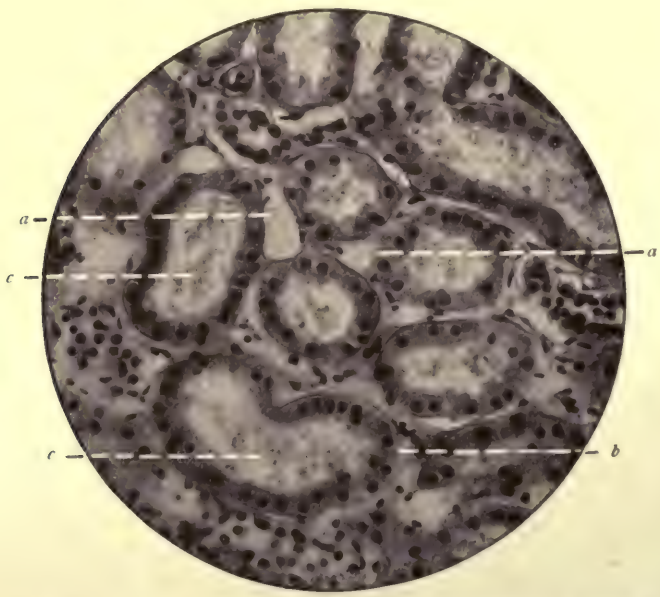
PLATE XI.

FIG. 1.



Acute Nephritis—cloudy swelling. *a*, Transverse Sections of Renal Tubules showing Lumen obliterated by Swollen and Granular Epithelium; Nuclei obscured; *b*, Inflammatory Exudate between Tubes. x 250.

FIG. 2.

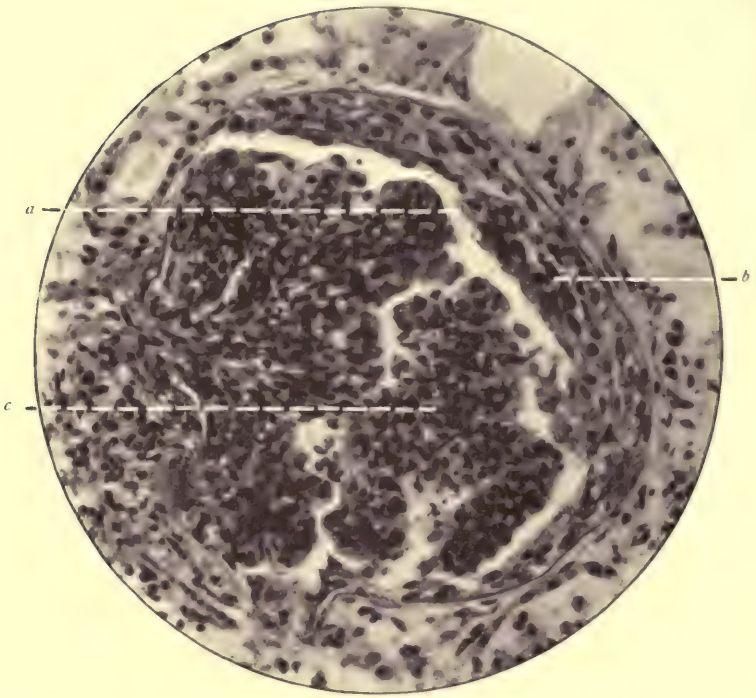


Acute Nephritis—Puerperal Convulsions. *a*, Dilated Capillaries; *b*, Granulo-fatty Degeneration of Tubular Epithelium; *c*, Lumen of Tubules filled with Granular Material. x 266.

Photographed by Henry S. Stearns, M. D.

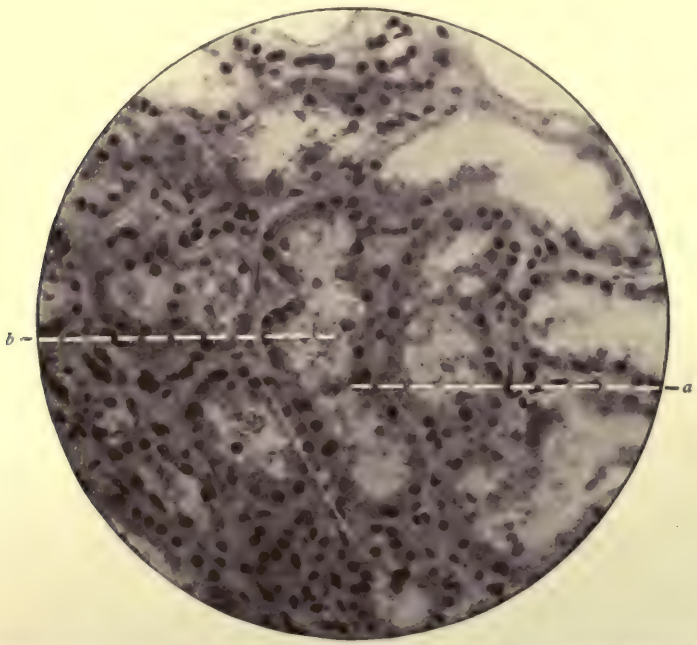
PLATE XII.

FIG. 1.



Acute Nephritis—Glomerulo-nephritis. *a*, Glomerulus; *b*, Capsular Space filled with Endothelium; *c*, Increase of Nuclei in Glomerulus. x 270.

FIG. 2.



Chronic Diffuse Parenchymatous Nephritis. *a*, Longitudinal Section of Convoluted Tubule showing Degenerated Epithelium; *b*, Lumen of Tubules filled with Granular Matter. x 210.

Photographed by Henry S. Stearns, M. D.

cases in which the urine was repeatedly examined before death and microscopical examinations of the kidneys were made after death, I found that in the 3 cases of acute nephritis (proved by autopsy) albumin and casts were always present during life, although the lesion was very slight. While this was not constant in the other kidneys of chronic nephritis, the writer believes that never are albumin and casts absent at any time in cases of acute nephritis.

In the cases of infectious disease accompanied by albuminuria, where death was due not to the kidney lesion, but to the primary disease, cloudy swelling and fatty degeneration of the cells of the convoluted tubes were found. When to this are added the evidences of inflammatory lesions, such as exudation between the tubes and changes in the glomeruli, then a true nephritis is developed. The alterations in the vessels of the glomeruli which allow albuminous fluid, white corpuscles, and sometimes blood to escape are the very earliest changes seen in acute nephritis.

Fatal Cases.—The following are the microscopical changes seen in the kidneys of fatal cases of acute nephritis (not all these changes are present in every kidney):

1. *Hemorrhages* into the uriniferous tubes, the capsule of Bowman, and between the tubes.

2. *Epithelium.*—Changes especially seen in the convoluted tubes, but not in all the tubes; generally in patches throughout each kidney: (*a*) cloudy swelling (see Plate XI. Fig. 1); (*b*) granulo-fatty change; (*c*) hyaline degeneration; (*d*) coagulation necrosis, especially seen in cases of poisonings.

Glomeruli (see Plate XII. Fig. 1).—(*a*) Space between tuft and capsule of Bowman filled with desquamated epithelium or emigrated white blood corpuscles; (*b*) cloudy swelling and granulo-fatty changes in epithelium covering tuft and lining capsules; (*c*) increased number of nuclei in tuft of vessels, often so extensive as to obliterate outline of vessels, due to a proliferation and desquamation of the endothelial cells of the vessels and a collection of white corpuscles in their lumen; (*d*) hyaline degeneration of the vessels of the glomeruli.

Tubes.—(*a*) Filled with desquamated renal epithelium; (*b*) granular matter derived from partly or completely broken down epithelium; (*c*) hyaline casts; (*d*) emigrated white blood corpuscles (see Plate XI. Fig. 2).

Intertubular Tissue.—(*a*) Often contains the ordinary products of inflammation, serum, leucocytes, and red blood corpuscles; (*b*) in severe cases round cell infiltration, especially about tufts, is seen; (*c*) desquamated endothelium and accumulation of white blood corpuscles fill capillaries.

Other Anatomical Changes found in those Dead from Acute Nephritis.—Serous cavities, especially the pleural, pericardial, and peritoneal, generally contain fluid; the two former show the evidences of a serofibrinous inflammation.

Lungs.—Lobar and lobular pneumonias common.

Brain.—Increase of the cerebro-spinal fluid and serous effusion in the meshes of the pia; sometimes meningitis is present.

Etiology.—All cases of acute nephritis are due to the direct local

irritation of the kidneys by toxic substances brought to them by the blood. Be this substance the ptomaïnes of an infectious disease, the product of an organic poison, or the excess of the waste products of the body, the resulting changes are the same, depending more on the amount and intensity of the poison than upon its character. What have been called different forms of acute nephritis are nothing else than distinct individual developments of the disease under its particular causes. With each individual case we have to describe the corresponding clinical and anatomical appearances.

Acute Nephritis after Infectious Diseases.—Acute nephritis may develop with any of the infectious diseases, and is due to the toxin of the disease acting directly on the vessel walls and epithelial cells of the kidneys.

In those diseases proved to be of bacillary origin it has been demonstrated by a number of observers that it is not the bacilli of the disease, but their products, the toxins, carried directly to the kidneys, which cause the trouble. For example, Roux and Yersin in the large number of cases of diphtheria examined by them after death have never found the Klebs-Löffler bacilli in the kidneys, nor when the bacilli were injected directly into the renal artery have they caused a nephritis; but intravenous injections of bouillon culture free from bacilli have always produced typical diphtheritic nephritis. The frequency of the occurrence of renal complications during the course of any of the infectious diseases depends on the natural mode of elimination of the poison of that particular disease. When other channels than the kidneys are used the complicating renal changes are rare or slight, as after typhoid fever kidney complications are rare, for the poison is eliminated by the fæces, while in scarlet fever the poison, being almost entirely eliminated by the kidneys, the excretion by the skin being temporarily stopped, acute nephritis is very common.

The amount of poison is an important factor in the development of an acute nephritis, and it is a well known fact that this form generally develops when the primary disease is at its maximum.

It has been observed that when the cause of the nephritis was endemic the resulting character and intensity of the nephritis did not correspond to the endemic disease; thus if the disease was severe the nephritis was not necessarily so, and if the endemic disease was light the nephritis would more probably be severe; this is especially seen in scarlet fever.

Scarlet Fever.—One of the most common causes of acute nephritis is scarlet fever. Some observers go so far as to say that one half of all the cases of acute nephritis are due to this primary disease. In the experience of the writer this statement, however, has not been borne out.

The occurrence or intensity of the kidney inflammation bears no relation to the severity of the fever; for example, mild cases of scarlet fever may be complicated by the most severe forms of acute nephritis, and, again, in the severest cases of scarlet fever the kidneys may not be affected.

The renal complications occur in the third or fourth week of the disease, during the period of desquamation. Often casts can be de-

tected during a period of high fever and before the presence of albumin. The finding of these casts in the urine, generally associated with white and red corpuscles, often enables one to foretell the development of kidney complications days before the albumin and clinical symptoms render the diagnosis certain. Scarlet fever developing in males is more apt to be complicated by an acute nephritis than that developing in females; also with the first the mortality is higher. Different years show different death rates.

In the fever hospital in London, Goodak saw 5443 cases of scarlet fever in ten years, and his conclusion as to the age at which concurring nephritis occurred was that the majority of cases was between the ages of five and ten, more rarely between twenty and forty, and after forty he has never seen a case. The mortality of all the nephritis cases due to scarlet fever he found to be—males 11.3 per cent., females 6.5 per cent.—and to be highest in cases under five years, the ratio at this age being 18.64 per cent.

Diphtheria.—There is a marked difference in the development of an acute nephritis during diphtheria and during scarlet fever. In the first disease, in the first place, diphtheritic nephritis appears especially in severe cases; it is estimated that 88 per cent. of the cases exhibiting the gangrenous form have the kidneys involved. In the second place, the acute nephritis develops during the height of the disease, so the early appearance of albuminuria with casts makes an early differential diagnosis possible.

The presence of albumin in the urine during diphtheria does not always indicate acute nephritis. Empis and Bouchet found albumin in two thirds of their cases. It generally appears on the third day of the disease, and is due as often to the disturbed circulation and the high fever as to the action of the toxins of the Klebs-Löffler bacilli. It has been found that there is no relation between the severity of the diphtheria and the presence of albumin, and that the albumin is accompanied by no clinical symptoms of acute nephritis in these cases. Acute nephritis complicates diphtheria much oftener than it does scarlet fever.

Hoppe-Seyler in Quincke's clinic found during the winter of 1889–90, among 455 cases of diphtheria, 238 cases in which an acute nephritis developed, or about 52 per cent.—17 per cent. of which were among adults and 61 per cent. among children. At the Willard Parker Hospital during the first eight months of 1895, 124 children were treated for diphtheria. In 2.4 per cent. of these cases death was attributable to an acute nephritis.

Typhoid Fever.—Acute nephritis as a complication of typhoid fever is not common, its frequency varying in different epidemics and under different lines of treatment. It usually occurs in the third to the fifth week of the disease, and is generally recovered from. Even cases with very bad urinary symptoms, such as a large amount of albumin and casts, make rapid recoveries.

Cases of typhoid fever are sometimes seen in which the urinary symptoms, coming on early and in a severe form, mask the ordinary picture of the disease. This form has been called by French and German writers the renal form of typhoid fever, and only the continued

high fever enables one to make a diagnosis, for acute nephritis as a complication has not a high fever. An acute hemorrhagic nephritis accompanied by functional disturbances, such as bloody urine, casts, etc., is sometimes seen in typhoid fever. It generally comes on early in the disease, and often proves fatal. The few cases which I have examined post-mortem showed scarcely any structural changes in the kidney, even under microscopical examination. Pneumonia, uræmia, and inflammation of the serous membranes are especially liable to occur during the course of an acute nephritis which complicates typhoid fever.

From July 1 to Nov. 1, 1895, during the writer's service as visiting physician to the New York Hospital, careful records were kept of the condition of the kidneys, as evidenced by the almost daily examination of the urine, of all the cases of typhoid fever under treatment. The especial object was to ascertain in how many of the cases the kidneys were involved, how severe and how permanent were the renal changes. Most of the typhoid cases were treated by the cold bath method, a few by the cold coil. The following are the tabulated results: Number of cases of typhoid fever, 54; number of cases with kidney involvement, 19. In 10 cases the urine contained albumin alone (the amount varying from a trace to 30 per cent.). In 2 cases the urine contained casts only (granular and hyaline). In 7 cases the urine showed both albumin and casts (from a trace to 40 per cent.). In 13 cases the albumin and casts appeared and disappeared. In 6 cases they did not disappear after convalescence (in only 1 case was there the previous history of nephritis); 6 of the 19 cases of typhoid fever with kidney involvement were mild cases; 13 were severe.

It was found that the albumin and casts appeared at the height of the disease, also that the amount of albumin lessened or disappeared as convalescence became established.

Intermittent Fever.—Some authors claim that malarial fever never causes acute nephritis. While I believe that in the northern part of this country it is one of the rarer causes, still in the malarial districts of the Southern States it is undoubtedly a very common cause. The writer has himself seen a case of true nephritis develop in Bellevue Hospital in a sailor who had just returned from Panama suffering with Chagres fever. The autopsy showed kidneys almost identical in appearance to those seen in scarlet fever, and in addition revealed extensive hemorrhages between the tufts and tubes. A distinct hemorrhagic form has been described by observers in Algeria, who also state that an acute nephritis is most apt to develop in those cases of daily fever accompanied by no sweating. The appearance of hemorrhages in the skin generally precedes that of hemorrhagic urine.

I showed lately at my clinic at Bellevue a young man with all the symptoms of acute parenchymatous nephritis (large white kidney), who was passing bloody urine containing albumin and casts. He had lived for a long time in an excessively malarial district, and under observation intermittent attacks of fever preceded by chills had been an accompaniment of his renal symptoms. On being treated with quinine all the renal symptoms quickly disappeared.

Erysipelas.—Acute nephritis as an accompaniment of erysipelas is

rare. A study of the records of the erysipelas pavilion of Bellevue Hospital shows—(1) that only 3.6 per cent. of the cases present renal complications; (2) that the acute nephritis appears early in the disease, begins to disappear as the primary disease declines, and finally all renal symptoms have disappeared by the time the patient leaves the hospital; (3) that the acute nephritis is generally of the hemorrhagic variety; (4) that the complicating nephritis does not correspond to the severity of the erysipelas.

The writer has seen at autopsy 2 cases of chronic nephritis which apparently had their beginning in an attack of erysipelas.

Pneumonia.—The writer's experience has been that albuminuria occurs in almost all cases of pneumonia, though this is not always due to nephritis. Neither is it due to the high fever, for albuminuria generally appears on the third or fourth day of the disease, and has no direct relation to the temperature. The chief factor is the infection, and the direct cause the secretion of the pneumonic poisons by the kidneys. The acute nephritis which follows pneumonia is generally mild, and almost all cases recover. It is a rare complication. In the 150 cases of pneumonia reported by Wagner only 4 had acute nephritis. If cases of chronic nephritis have pneumonia, there is almost always an acute exacerbation of the nephritis.

Acute nephritis developing during the course of an acute pneumonia has no influence on the pulmonary trouble, except that the day of crisis is delayed. Symptoms of the nephritis last six to eight weeks after recovery from the pneumonia has occurred.

Hemorrhagic forms of acute nephritis sometimes occur with lobar pneumonia. Causard reports 48 cases of this variety.

Smallpox.—Acute nephritis with smallpox is seldom seen. It sometimes occurs in the stage of eruption, especially at the time of the eruption of the pustules—from the sixth to the eighth day. Blood is often present in the urine of those suffering from smallpox, but it comes primarily from the pelvis of the kidney and not from the parenchyma.

There may be severe and even fatal disease of the kidneys in such a mild disease as varicella.

Acute Articular Rheumatism.—Acute nephritis is very seldom seen as an accompaniment of acute rheumatism. Even albuminuria is rare. It is a question whether nephritis is ever present unless there is a complicating endocarditis. In the Zurich clinic among 360 cases of acute rheumatism there were only 4 of acute nephritis. Fürbringer found in 1000 cases only 5 of acute nephritis. Cases have been reported where the acute nephritis of acute rheumatism has passed into the chronic form. Bartels reports 2 such cases. An acute nephritis may develop in the course of a chronic articular rheumatism.

Yellow Fever.—Yellow fever is sometimes followed by an acute nephritis, but the frequency of the complication varies with different epidemics. From the number of specimens of different kidneys sent to the writer by the health officer having charge of the epidemic in Florida a number of years ago, he would judge that acute nephritis is not an uncommon complication. The most prominent change in the kidneys was an acute fatty degeneration of the cells, accompanied by change in the intertubular tissue.

Traumatism and Suppuration.—After simple fractures of long bones albumin, blood, epithelium, and casts, especially the hyaline variety, sometimes appear in the urine, and the symptoms of acute nephritis develop. The reason for the development of this form of nephritis has never been satisfactorily explained. It is, however, generally curable. Sometimes acute nephritis is established in cases of prolonged suppuration due to the elimination of the septic poisons. The changes in the urine may often be the only signs of this affection. The changes in the kidney which accompany the development of septic abscesses—miliary embolic abscesses—cannot be classified under the head of acute nephritis.

Syphilis.—That the poison of constitutional syphilis may act as an exciting cause of an acute nephritis is known. Baumberger says that in 8 per cent. of syphilitic cases there is a mild form of desquamative nephritis during the eruptive stage of the disease. Acute nephritis is not often seen with syphilis. When it is so seen it has no characteristic symptoms, and is especially benefited by mercury.

Pulmonary Tuberculosis.—Here the acute nephritis is due to the absorption of septic products from the excavations in the lungs and from the caseous pneumonic processes. The nephritis develops late in the disease. The writer has seen improvement in phthisis on the development of an acute nephritis. Albumin appears temporarily in the urine in a third of all cases of phthisis.

Nephritis after Exposure to Cold.—The writer does not believe that exposure to cold alone will cause nephritis. Rabbits were shaved and exposed to hot and cold water, but no nephritis developed. If the secretion by the skin is interfered with, as by chilling of the surface and lowering the vitality, and *at the same time* there is excrementitious material in the blood, the elimination of this is suddenly thrown on the kidneys, and an inflammation of these organs may result. It is not the cold, *per se*, that causes the trouble, but the cold plus a certain blood condition. It is a well known fact that acute nephritis often develops in those who expose themselves to cold while under the influence of alcohol. Is it not the blood condition induced by the prolonged use of alcohol that turns the balance in these cases, and not the cold? If the history of those cases in which acute nephritis apparently develops from exposure to cold was carefully examined, it would be found that either the persons were physically below par, as from fatigue, depressed nervous system, etc., or they had been absorbing alcohol for a long time and their blood was loaded with the product of tissue waste. This has been the writer's experience in a large number of cases whose history he carefully examined. Many cases of acute nephritis which are supposed to be due to cold are really due to an infectious cause. Moist cold and sudden exposure are especially dangerous.

Toxic Nephritis.—Our knowledge of toxic nephritis from poisons is the most extensive, for it is a form that can be produced by experiment. There are medicines which act on the kidneys as diuretics and in overdoses have a toxic effect, such as cantharides. Careful methods of medication have reduced the frequency of nephritis from poisonous causes. Acute nephritis has been developed by the internal use of cantharides, turpentine, squills, and chlorate of potash; also by the ab-

sorption of carbolic acid, bichloride of mercury, balsam of Peru, and possibly iodoform. When applied locally to large absorbing surfaces, the mineral acids, arsenic, phosphorus, and lead do not cause a true inflammation of the kidney, but produce degenerative changes, especially fatty. Cantharides affects both the parenchyma and interstitial tissue of the kidney and causes albumin, red blood corpuscles, and casts of various kinds to shortly appear in the urine. In severe cases death occurs early in the disease, while in mild cases recovery soon follows. Chlorate of potash in large doses causes changes in the blood, destruction of the red blood corpuscles, etc., and finally a true inflammatory change in the kidney with very dark scanty urine containing albumin, degenerated blood cells, and casts of broken-down epithelium. Acute poisoning by bichloride of mercury and carbolic acid produces more of a degeneration than an inflammation of the kidney. In a number of cases examined by the writer he has found the prominent change to be coagulation necrosis of the epithelium of the kidney. Certain substances, as bile salts or salicylate of soda, may irritate the kidneys without producing an inflammation. It must be remembered that some persons have kidney tissue which is non-resistant to almost any irritation.

It is not a settled fact whether alcohol will produce nephritis in man. Renzold has certainly produced a fatal nephritis in dogs by giving them alcohol.

Pregnancy.—This form of nephritis was first recognized in 1842. At first it was supposed to be due to the pressure of the gravid uterus on the renal vessels. Now it is believed that the hydræmic condition of the blood, loaded as it is with an excessive amount of waste material, causes the inflammatory changes, which are confined to the epithelial cells of the convoluted tubules. There are no glomerular or interstitial changes. (See Plate XI. Fig. 2.)

Pregnancy does not necessarily increase a previous kidney disease. The rapid recovery after confinement is the best proof that the kidney lesion is due to the pregnancy.

Virchow lately has called attention to fat embolism in the kidneys and the lungs in connection with the gravid uterus. Leyden holds the view that the nephritis of pregnancy is due to arterial anæmia combined with fatty degeneration, this being produced either by a swelling in the cortex or a stasis in the urinary tubules. The few kidneys which the author has been able to examine were anæmic, of a pale yellow color, softer and larger than normal. The microscope showed changes confined entirely to the epithelial elements of a granular and fatty character.

The nephritis of pregnancy occurs in the second half of pregnancy, never before the third month. It occurs especially in young women and those bearing twins. The symptoms are insidious in their appearance. The urine is generally diminished, of high specific gravity, and contains a considerable quantity of albumin; hyaline casts are always, and granular casts generally, found. Most of the symptoms found in acute nephritis are present, but in a mild degree. The possibility of the sudden development of uræmic symptoms is always before the patient.

Symptoms.—*Advent Sudden.*—This is liable to occur when the nephritis is developed by a sudden cold or an acute exanthem, such as scarlet fever. The patient has one or more chills, followed by fever,

pain in the back, which is increased by deep pressure, and general malaise. The urine becomes scanty and high colored; œdema develops, and uræmic symptoms, marked by constant vomiting and convulsions, quickly supervene. These cases of sudden advent are the rarer forms. Usually the disease is ushered in as follows:

Advent Slow.—In these cases we can often only tell the beginning of the process by the dark colored, scanty urine. The symptoms may be so mild that the first noticed are the uræmic ones. Generally, after a few days, there is a little œdema, especially of the eyelids. At first this is seen only in the morning, and passes away in a few hours, but later it becomes more permanent and general. Frequently the œdema is the first sign. As the disease advances the digestive organs are disturbed; nausea passes into persistent vomiting; constipation gives place to diarrhœa and causes loss of strength. Weakness, persistent headache, pains in the back and loins, swelling of the feet, pallor of the face, dryness of the skin, and sometimes dyspnœa, are prominent early symptoms. The urine is soon greatly diminished in quantity, the patient often passing only ten or fifteen ounces in the twenty-four hours. Fever is not a symptom of acute nephritis, and when present is generally due to the primary condition which caused the nephritis.

If the disease becomes arrested, the fever, when present, subsides, the dry skin becomes moist, the œdema disappears, the nausea and headache subside, the urine becomes more profuse and lighter in color, and gradually the patient recovers. This is the termination in nine tenths of the cases of acute nephritis. (See classification, Nephritis, page 706.) In a few cases the albuminuria and œdema do not disappear entirely; the patient presents a pale waxy look, is troubled with weakness and shortness of breath; in this way the acute form of the disease merges into the chronic. In other cases which pass to a fatal termination all the symptoms increase in severity; the urine is scanty and remains of immense quantity, and to this are added nervous symptoms, constant severe headache, convulsions, which are of epileptic form and character, to be followed by stupor; and these symptoms continue constantly to the end. Sometimes these attacks do not occur at all, but complications, such as œdema of the lungs, or pericarditis, may end life.

Such may be the three terminations of all cases of acute nephritis—recovery, death, or the development of chronic nephritis. Owing to the diseased organs or to the effects upon the system of the retained body poisons not eliminated by the kidneys, there are certain symptoms which will now be described in detail.

A positive diagnosis of acute nephritis can only be made by means of the urine.

The Urine.—The quantity of urine is always diminished, sometimes to 100 c.c. in twenty-four hours, and cases have been reported with recovery where only 50 c.c. were passed daily for a number of consecutive days. The color of the urine is generally dark red and turbid. Urine containing blood in quantities gives a greenish color when held before the light. Urine passed during the night contains less blood than that passed during the day. The urine generally shows upon standing a red brown colored sediment. The specific gravity is high, 1020 to 1030. Serum albumin is present in large quantities, ½ to 1 per cent.;

5 to 25 gr. are passed daily. The urea is reduced to one sixth or less of the normal amount. The phosphates and chlorides are reduced. A microscopical examination shows fine hyaline and epithelial casts, lymphoid cells, kidney epithelium, and granular matter. Often there are crystals of oxalate of lime or uric acid hæmatoidin free or in cylinders and micro-organisms. The largest number of cellular elements is found in the urine of scarlet fever and diphtheria.

There are cases of acute nephritis where the cell elements are not found, but only blood casts. In many cases the casts appear in the urine before the albumin, and may be found after the albumin has disappeared. At different times of the day the number of casts vary, though the amount of albumin is constant. Casts are not always found in the urine in acute nephritis. Sehrwald has proved the solubility of casts due to the pepsin found in the acid urine, and has also proved that the longer the urine remains in the bladder the fewer the number of casts found, and also the higher the temperature the fewer casts. The disappearance of albumin from the urine is not an infallible sign that the disease has run its course.

Dropsy.—The symptom of dropsy is next to the urine in diagnostic importance. Dropsy begins in the eyelids, then appears about the ankle and along the tibia, and finally extends up the limbs. The transparent swelling of the subconjunctival tissue, the pale red cheeks,—these constitute a peculiar physiognomy which is easily diagnosed. As dropsy becomes extensive the penis and scrotum become œdematous and fluid collects in the serous cavities. The most important dropsy is that of the lungs, brain, and epiglottis, the latter especially being often the cause of sudden death.

As dropsy is affected by the diminished amount of urine and sweat, this may account for the severe dropsy after exposure to cold and after scarlet fever, while in some infectious diseases there is little or no dropsy, even in otherwise very severe and fatal cases.

Teichlenstein has proved that these œdemas may be of an inflammatory nature. He has observed a serous lobular pneumonia.

The skin becomes dry and harsh; there is but little tendency to sweating. It is pale, shining, without wrinkles, and pits on pressure. Anasarca may be so extensive as to cause fissures in the skin, and infection thus arises. It may be the cause of erysipelas in the same way, or even gangrene.

The Pulse.—During the greater part of the course of an acute nephritis the pulse is not accelerated. In some rare cases it is very slow (36 to 48). It is often not possible to detect any changes in the pulse in the ordinary way, but the sphygmograph will reveal a high tension in the vessels. Hypertrophy of the left ventricle is seldom seen. Sometimes after two or three weeks there will be evidences of slight dilatation of the left ventricle. The second aortic sound is often increased.

The Blood.—The specific gravity of the blood varies from 1018 to 1024. The water is increased. Frerichs found that albumin in the serum, instead of being from 69 to 79 per thousand, was only 51.7. The most marked change, however, is the amount of excrementitious material present, sometimes so great that the skin will be discolored.

Gastric Symptoms.—Nausea, vomiting, and diarrhœa are common

symptoms; the latter, however, is most commonly seen in chronic forms. Vomiting, which appears as one of the ushering-in symptoms of acute nephritis, is either reflex or due to diminished diuresis, and is one of the signs of uræmic poisoning.

Nervous Symptoms.—These symptoms are due to uræmia. They are headache, apathy, restlessness, dyspnœa, asthmatic attacks, and muscular tremors, the latter due to cortical irritation. The nervous phenomena may be followed by epileptic convulsions, which are repeated until finally coma or death closes the scene; or they may grow shorter and less severe until sweating and an increased flow of urine gives relief to the kidneys, and the patient recovers. Children and nervous persons are especially liable to have these nervous symptoms in a very pronounced way.

Diagnosis.—It is often with the greatest difficulty that a diagnosis can be made between an *acute nephritis* and *an acute exacerbation of a chronic nephritis*. I have been impressed with this fact during my service as curator of Bellevue Hospital. In the course of a year a large number of cases with the diagnosis of acute nephritis came to the dead house, the diagnosis often made by some of the best diagnosticians in the city. In the great majority of cases the autopsy would show the characteristic kidneys of a chronic nephritis. The following are the points to be considered in the differential diagnosis of these conditions: (1) *history of the case*, noting especially the day of the first symptoms; (2) *the general appearance of the patient*, as in the chronic form with exacerbation there is an appearance of longer illness, pallor, swelling of the skin, etc.; (3) in acute nephritis the specific gravity of the urine is higher and it usually contains blood; (4) if the case is one of chronic nephritis with acute exacerbation, there will be evidences of cardiac hypertrophy, high arterial tension, and retinitis albuminurica.

It is sometimes difficult to determine whether an *albuminuria is febrile or due to an acute nephritis*. The history of the case and the microscopical examination of the urine will aid in determining this point. When the nephritis and fever appear together, the nephritis should be considered as following the fever—fever not being a symptom of acute nephritis.

Prognosis.—Acute nephritis may end in complete recovery, incomplete recovery marked by development of chronic nephritis, or in death. The plurality of cases recover, a few develop chronic nephritis, and only seldom is death a final result. Prognosis is influenced by the cause, the condition of the patient when attacked, and the intensity of the symptoms. Scarlatina and malarial nephritis show a special tendency to become chronic; also nephritis complicating diphtheria, suppuration, and burns. The nephritis of pneumonia, typhoid fever, and that due to a cold, although very severe, is generally recovered from.

Of any form of nephritis, that accompanying scarlet fever is least to be trusted. The following symptoms render the prognosis unfavorable: Pulse: small, frequent, and soft, with or without cardiac dilatation. Urine: the amount passed daily is one of the best guides we have as to the prognosis of the case; complete or nearly complete suppression is unfavorable. Uremia: if accompanied by convulsions or coma.

Dropsy: if excessive. Inflammatory complications, such as œdema of the lungs, pharynx, and fluid in the serous cavities.

Duration.—Favorable cases generally recover within four weeks. Many, however, are protracted to many weeks, even months, and still recover. Of these longer cases, six months is the average time before recovery is completely established. As long as the urine contains casts, even if no albumin be present, the disease is not cured. Fatal cases almost invariably die early in the disease.

Treatment.—In the treatment of acute nephritis it must be remembered that the majority of cases recover if carefully managed, and in no disease is attention to hygiene and diet more important. Rest in bed, protection from changes in temperature, and regulation of diet will cure most cases without medicine, but care must be constantly exercised until convalescence is fully established. One of the first points to decide in the treatment is whether the disease causing the nephritis is present and still acting. If so, the primary condition should be as carefully attended to as the resulting nephritis.

Treatment of acute nephritis may be considered under the following heads:

I. **Hygienic Regulations.**—As soon as acute nephritis is diagnosed, and even during the course of those diseases in which it is especially liable to develop, attention to certain practical details is imperative.

It is always necessary for the patient to remain in bed and wear woollen in contact with the skin. This may often be facilitated by having woollen blankets used to cover the patient and for him to lie upon. The temperature of the room should be kept between 68° and 72° F., and care should be taken that the room is thoroughly ventilated. The skin should be kept in good condition by daily spongings with tepid water, followed by gentle but rapid friction. Inunctions of oil to the surface of the body do good. They act to prevent evaporation—force the water through the kidneys, which acts as a diuretic. All baths should be interdicted, for it must be remembered that it only takes a fraction of a minute to take cold after a warm bath, and that uræmia has been known to suddenly develop after a bath.

Diet.—In the treatment of nephritis alimentary hygiene is of the greatest importance. This constitutes the real treatment, and medication is of secondary importance. This should be impressed upon the patient, and also that every imprudence and every violation of the rules laid down may result in grave consequences, even to the endangering of life itself.

Milk is the food par excellence, and during the acute or subacute stage it should be the only diet. Three quarts in twenty-four hours are sufficient, and can be easily borne by the patient if taken in goblet-ful quantities every two hours and drunk slowly. Seltzer or Vichy water may be added to the milk to make it more palatable. If the milk causes distress and is not digested readily, peptonized milk, buttermilk, or koumyss may be substituted. As a prophylaxis in scarlet fever a milk diet should be exclusively used. It has been proved that in 100 cases of scarlet fever nephritis was prevented in 97 by this treatment.

In the later stages of those cases of acute nephritis which are tending toward recovery convalescence may be facilitated by discontinuing

the exclusive milk diet, which has often become by this time very distasteful to the patient. The milk diet is not only not necessary now, but may be harmful—(1) because the patient's stomach cannot digest it; (2) because the albumin will not be replaced by the hydrocarbons; and (3) because the decreased amount of albumin will not be able to nourish the patient.

At this time the following articles of food may be given: meat soup, veal, chicken, or beefsteak, fish, bread, potatoes (finely divided), soft-boiled eggs (sparingly), sauce, pastry, farina, prepared milk, quantities of red or white wine, with water, weak tea, cream, fresh cheese, and alkaline mineral waters. If there are any uræmic symptoms, no meat should be allowed. At the least menace there should be no hesitancy in returning to the simple milk diet. When there is a relative recovery the patient may return to the mixed diet with vegetables, but it should be more or less restricted for a long time.

Bowels.—The first thing to do after prescribing a milk diet is to relieve the system of toxins, and for this the best method is by the bowels. The very best eliminative, in my opinion, is calomel, and this given in minute doses, as one tenth of a grain often repeated. This will at once increase the flow of urine, relieve any uræmic symptoms, and prevent toxæmia. In the treatment of this disease, of all drugs, I should place calomel first. Its action is often increased by afterward giving a saline, especially if there be any tendency to constipation.

Skin.—After the bowels, the skin is the channel which must be utilized to remove the toxins from the system and so relieve the kidneys. If there are symptoms of uræmia and much œdema, diaphoresis should be employed. The best method is by hot packs. The patient should be wrapped in hot blankets wrung out in water as hot as can be borne by the hand. Over these should be placed dry blankets to keep in the heat. To increase the sweating, hot water bottles should be laid alongside the patient and warm drinks should be given.

The advantages of hot packs over the warm bath are—(1) they are not so depressing; (2) there is less exposure and consequent danger of taking cold; (3) they may be repeated oftener.

With robust patients, suffering with acute symptoms and rapidly developing uræmia, the warm bath may be given. The following is the method as recommended by Liebermeister: The temperature of the bath should be 37° C. when the patient enters it, and then run up to 40° or 43° C. At the same time cold should be applied to the head to relieve any congestion. The bath should last from fifteen to thirty minutes, and after the bath the patient should be wrapped in warm blankets and covered well. At first the bath can be given every second day, and afterward once or twice each day. The best results are obtained only after two or three baths. Diaphoresis is due to reflex irritation of the nerves of the skin. In certain cases, especially those too weak to bear the exposure and moving necessary in the above methods, hot air may be introduced under the bedclothes either by an especially constructed apparatus or by a protected spirit lamp, the bedclothes having been raised and supported.

The hot air bath is used much more in this country than in England or on the Continent. While the writer favors the first method—hot

packs—he still believes the hot air bath will give the best results in a limited class of cases.

Marked cardiac weakness, prostration of the patient, great dyspnœa, fever, and inflammatory complications are contraindicated in the employment of any of the above methods.

In cases where the urine is at a minimum, urgent uræmic symptoms are developing, œdema of the lungs is threatening, and hydro-thorax and ascites are interfering with circulation and respiration, drugs are indicated.

II. **Medicinal Treatment.**—To relieve the congestion of the kidneys and to excite the flow of urine, especially when it is rapidly diminishing in quantity, dry cups over the lumbar region should be resorted to before any diuretic drugs are given. The action of the cups may be intensified by applying poultices made of digitalis leaves after the cupping.

In the selection of diuretics great care should be exercised, and only the milder ones employed. Liquor ammonii acetatis in teaspoonful doses every four hours or in larger doses three times a day has given me the best results. Citrate of potash, two to three drachms per day, is often effective; also benzoate of soda.

Diuretin, one to two drachms a day, has been lately recommended very highly by German observers. It does not contract the lumen of the kidney vessels. In cases where the writer has tried it the results have been very satisfactory. It should be given in solution with peppermint water after meals. The very best diuretic is water, and the patient should be encouraged to drink as much of it as possible. Cream of tartar, ʒj to a pint of water to which half a lemon and a little sugar have been added, makes a very refreshing drink. Only in rare and desperate cases is digitalis indicated, to increase the tension of the renal vessels and so produce diuresis. The indiscriminate use of this drug does more harm than good. When the action of the digitalis on the kidneys is desired, the infusion should be used, combined with the bitartrate or acetate of potash. (The infusion should be made from the fresh leaves and not from the fluid extract.) If the digitalis does not at once increase the flow of urine, it should be stopped.

Heart.—The action of the heart should be watched in every stage of the disease. If there is evidence of cardiac failure, a mild cardiac tonic may be used, as tincture of strophanthus or the infusion of digitalis in small doses. For a high tension pulse chloral hydrate, five grains every four hours, should be given.

Vomiting.—If vomiting is constant and cannot be controlled by the use of peptonized milk or koumyss, iced champagne will generally relieve it. Minute doses of calomel combined with bismuth will often prove effective if any medication is indicated.

Bowels.—If constipation is present, the bowels may be kept open by sulphate of magnesia, ʒij in a glass of water early in the morning, or in ʒj-doses repeated during the day.

Uræmia.—In spite of all care, uræmia may set in. When the first symptoms show themselves attention should be given to the bowels, and one of the mild hydragogue cathartics administered. Elaterium acts promptly, but it should be used with great care. The compound jalap

powder is safer. In strong patients, if the pulse is full and the face congested and cyanotic, bleeding should be resorted to. The writer has seen it work admirably, but he believes it to be seldom necessary. For uræmic headaches apply ice bags and give small doses of antipyrine, caffeine, or nitro-glycerin. For uræmic convulsions use chloroform even to narcosis. Morphine hypodermically may also be used. Other drugs, such as chloral, bromide, or oxygen, are not effective. Urethrane in solution, up to one hundred grains in twenty-four hours, has given the writer good results.

Dropsy of the skin and serous cavities requires mechanical interference, and the sooner this is done the better for the patient. Often in the beginning the dropsy can be controlled by massage and bandages to the legs. Œdema of the epiglottis requires quick action. The tumor should be scarified, and if this is not effective and death threatens, tracheotomy should be performed.

During convalescence gentle exercise may be allowed, such as walking or riding, if the patient is able to take it, but never to the point of fatigue, and then always with precautions against cold.

In case of anorexia tonics, hydrochloric acid, etc., may be given. Alcohol and tobacco should not be allowed, and men should practise the greatest sobriety in venereal indulgence. Women should not marry for a number of months after complete recovery.

Chronic Diffuse Parenchymatous Nephritis.

Synonyms.—Chronic diffuse parenchymatous nephritis; Chronic tubal nephritis; Second stage of Bright's disease; Chronic desquamative nephritis; "Large white kidney;" Chronic glomerulo-nephritis.

This is the most common form of nephritis, and, as in this form certain distinct morbid conditions of the kidney are associated with characteristic clinical phenomena, the disease may be considered under three heads. There are *three varieties of kidneys* found at the autopsy, differing distinctly one from the other both as to their gross and as to their microscopical appearances. In all three varieties the primary changes begin in the epithelial cells of the organ (a parenchymatous inflammation).

The mode of development and the symptoms presented by these kidneys enable the clinician to foretell accurately what kind of kidneys will be found at the autopsy; therefore the description of each kidney and the symptoms attending it will be considered separately, while the etiology and treatment will be embraced under one head.

Forms of chronic diffuse nephritis (based on the appearances of the kidneys):

(1) Large white kidney;
(2) Chronic hemorrhagic kidney;
(3) The mottled or secondary cirrhotic kidney (smooth cirrhotic).

Pathological Anatomy.—(1) **Large White Kidney.**—This kidney is found under two conditions: (1) where acute nephritis has passed into the chronic form; (2) where the disease is chronic in form from the beginning. This form of nephritis is not a common one. With it the patient generally dies within two years of the development of the dis-

ease. The large white kidney may sometimes, owing to the secondary development of connective tissue, change into the mottled or smooth cirrhotic kidney.

Gross Appearance.—The kidneys are always enlarged, their surfaces smooth, and their capsules non-adherent. The color is generally pale, sometimes yellow or white, and mottled with red points on the surface (injected stellate veins).

On section the cortex is increased, of a yellowish white opaque color, dotted here and there with hemorrhagic and congested portions; the normal markings are obliterated; the medullary portion is darker than the cortex.

Microscopical Appearance.—Hemorrhages are seen within the capsule of Bowman and in the convoluted tubes, sometimes between the tubes. The epithelial cells lining most of the tubes will have undergone granular and fatty change. In some cases the cells will be atrophied and the tubes collapsed; in others the epithelium will appear normal. Glomeruli in certain areas will be shrunken and their capsule thickened by new connective tissue; in others the epithelium of the tuft and capsule will be altered and desquamated (glomerulo-nephritis). The vessels of some glomeruli will have undergone hyaline degeneration; others are fatty and blocked with white blood corpuscles. Connective tissue is increased—not generally throughout the cortex, but in circumscribed patches, especially around the tufts and the convoluted tubes in close proximity to the glomeruli. Never is the increase of connective tissue as great as in the mottled or secondary cirrhotic kidney. Renal tubes are filled with granular matter, the remains of broken-down cells. They often contain hyaline casts and red blood corpuscles. In some cases the tubes will be found entirely denuded and collapsed, and in other places they will have disappeared and their place will be taken by new connective tissue (Plate XII. Fig. 2.)

(2) **Chronic Hemorrhagic Kidney.**—*Gross Appearance.*—The kidney is enlarged, pale, smooth, and hard. On section the cortex seems to be studded with red and brown patches of hemorrhagic extravasation.

Microscopical examination shows extensive hemorrhages in the capsule of Bowman and in and about the convoluted tubes. The interstitial tissue is infiltrated in very many cases with round cells, and areas of beginning cirrhosis are apparent. The epithelium of the uriniferous tubules shows a considerable degree of fatty degeneration. In some tufts the epithelium of the glomeruli is desquamated. Often this kidney presents most of the changes found in the large white kidney, but the extensive hemorrhages make it a characteristic form readily diagnosed at autopsy. The symptoms during life enable it to be differentiated, and, while I believe it to be the rarest of all forms of nephritis, to make any classification complete it should be described as a distinct variety.

(3) **The Mottled or Secondary Cirrhotic Kidney.**—This is by far the most common kidney of nephritis met with at post-mortem examinations; 20 out of 46 kidneys showing the lesions of acute or chronic nephritis, examined microscopically by the writer, belonged to this class. It will be found to be the most common variety of nephritis

when the diagnosis is based on an accurate microscopical examination. This kidney shows, in connection with the degeneration of the epithelial element, a gradual increase in the connective tissue of the organ and a consequent shrinkage. The two changes seem to develop slowly and together; as the epithelium degenerates and the tubes collapse new connective tissue fills in the areas.

Gross Appearance.—The kidneys are generally a little larger than normal, rarely smaller; the consistency is firm, the color is reddish and mottled with gray patches, the red places corresponding to the new connective tissue bands extending inward from the capsule, the gray or yellow areas to the elevated portions made up of degenerated tubes. The surface is slightly uneven and the capsule in places adherent, but still easily detached. The large size of the kidneys, the only moderately adherent capsule, the coarseness of the granulations, readily distinguish these kidneys from those of chronic diffuse interstitial nephritis.

On section the appearances are more marked, the cortex is enlarged. Pale and gray striations are mingled with red ones, the medullary substance is darker than the cortex, and the columns of Bertini are prominent and have a mottled appearance.

Microscopically, the stroma between the tubes is increased by cell infiltration and new connective tissue. The glomeruli are extensively changed; some have capsules thickened by rich cell infiltrations which compress the tuft of vessels. With others the capsule is greatly thickened by concentric rings of new fibrous tissue. In some glomeruli the capillaries are distinct, in others the epithelium is destroyed in part or entirely. In many tufts the vessels have undergone hyaline degeneration. The epithelium of the glomeruli and tubes shows all the stages of granulo-fatty degeneration from moderate to complete destruction of the cells.

Arteries.—In the small and medium-sized arteries the media is often thickened.

Other Pathological Lesions.—*Heart.*—In this form of nephritis the heart is almost invariably found hypertrophied, the wall of the left ventricle being very much thickened and often the cavity is dilated.

Arteries.—In many cases the walls of the small arteries are found thickened, especially with the mottled or secondary cirrhotic kidney.

ETIOLOGY.—This form of nephritis is generally found between the ages of twenty and fifty, seldom if ever in early youth and old age; when seen in children it is always secondary to scarlatinal nephritis. Men are affected much more frequently than women. Heredity is an important factor in development. Dickinson reports 18 cases in three generations of the same family. Kidd had 7 cases in three generations of the same family.

Habitation and occupation have much to do with the development of this form of nephritis. It is a disease especially of the poorer classes. Those who dwell in unhealthy and damp buildings, who work amid antihygienic surroundings, and are exposed to changes of temperature are liable to it.

The most important etiological factor, in the writer's opinion, is alcoholism. Generally it is the persistent use of alcohol, especially of malt liquors, that causes it. It is the steady, every-day drinker who

may never be intoxicated who readily develops the disease. In 22 cases examined, where the diagnosis of this form of nephritis was made by a microscopical examination of the kidneys, the writer found a marked alcoholic history in 15, or nearly three fourths of the cases. A study of the hospital records at both Bellevue and the New York hospitals confirms this opinion of alcohol as being the great etiological factor, in this country at least, in the development of this form of nephritis.

It is not always easy to establish the etiology of chronic parenchymatous nephritis. The duration of the disease and any preceding diseases must be taken into account. When an acute nephritis has passed beyond eight months, it may be considered as chronic and that the large white kidney has developed. When secondary to an acute nephritis it generally follows pregnancy, or the scarlatinal or malarial form of nephritis.

Chronic malarial affection is often a cause; especially is this seen among those living in intensely malarial districts. Observers in Algeria have noticed this form of nephritis complicating malaria in a large number of cases.

Chronic endocarditis is sometimes a cause of this form of nephritis. Bamberger's statistics would indicate that 7 per cent. of the cases could be attributed to endocarditis and valvular lesions.

The other causes that sometimes produce it are phthisis, chronic suppurative processes, syphilis, and the chronic rheumatic poison.

Symptoms.—The three forms of this disease are differentiated clinically principally by the manner of their advent and the history of the case. Persons often consult a physician when the disease has been established for a long time and has been possibly overlooked. It is then only by a very careful inquiry into the history of the case that a diagnosis of the variety of this disease can be reached.

The points of difference between these three forms will first be discussed, and then the symptoms common to all the forms will be considered; for after this variety of chronic diffuse nephritis is once established the clinical picture of its different forms is practically the same. The hemorrhagic form is more interesting from an anatomical than from a clinical standpoint. It is rarely seen, and has only been introduced to make the picture of this variety of nephritis complete in all its details.

(1) **Large White Kidney.**—By this name are known the cases in which acute nephritis, instead of being recovered from, as it generally is, has continued for a number of months and chronic parenchymatous changes have become established. When the scanty, high colored urine of the acute condition gives place to free urination, often excessive in amount, and the acute symptoms, such as the large amount of albuminuria and dropsy, have subsided, then the history of the chronic process can be said to have begun. The dropsy, albuminuria, and anæmia never disappear, but begin again to develop slowly, and are hard to combat. The pale face and puffy eyes are characteristic. There is œdema of the feet. This form of parenchymatous nephritis is marked by the extensiveness and great tenacity of the œdema. The anæmia is marked. The heart is not hypertrophied. The urine is of high specific gravity, rich in albumin; often 20 grammes are passed daily:

certain appearances in the microscopical elements of the urine are characteristic.

(2) **Chronic Hemorrhagic Kidney.**—The symptoms which mark the development of this form are similar to those of the following variety. The hemorrhagic urine and the obstinate dropsy are the characteristic symptoms. No matter what careful treatment the patient may have, constant rest and good nourishment, and even the flow of urine increases up to the normal amount, yet the dropsy will not diminish.

Mild cardiac hypertrophy is present, and often signs of changes in the vessel walls are observed.

The other symptoms present may be similar to those of the mottled or secondary cirrhotic kidney.

A number of writers—among whom are Rosenstein and Aufrecht—do not consider this as a distinct form of chronic diffuse parenchymatous nephritis, but believe it is an acute hemorrhagic condition engrafted upon the chronic type.

(3) **The Mottled or Secondary Cirrhotic Kidney.**—These cases are chronic from the beginning. They often develop without any special symptoms. The first symptom which generally leads the patient to consult the physician is the dropsy, which is generally mild and seen first about the ankles and then on the eyelids. The patient is pale and complains of headache, fatigue, shortness of breath, and cardiac palpitation after exertion.

The changing intensity of the clinical picture and the extent of the dropsy, varying as it does from a slight amount about the ankles to general anasarca, is diagnostic of this form. These cases often, under careful medical care, apparently almost recover—all the subjective symptoms disappear. The constant presence of albumin, with generally a few casts and slight œdema at times, are the only marks that the disease still lingers. Relapses occur at any time, and the patient may have ups and downs for a number of years, until finally some complication closes the scene.

Chemical and Microscopical Condition of the Urine in Typical Cases of the Three Varieties of Kidneys seen in Chronic Diffuse Parenchymatous Nephritis.

Urine.	Large White Kidney.	Mottled or Secondary Cirrhotic Kidney.	Chronic Hemorrhagic Kidney.
	Chemical.		
Amount	400–1200 c.c.	600–1500 c.c.	2000–3000 c.c.
Color	Yellow, yellowish red.	Light yellow turbid.	1012 or lower.
Specific gravity	Above 1020.	Above 1016.	Reddish yellow.
Reaction	Acid.	Acid.	Acid.
Sediment	Present, turbid.	Present.	Present.
Urea	Diminished.	Diminished.	Diminished.
Albumin	Abundant, 1–5 per cent.	Fairly large, varies.	Abundant.
	Microscopical.		
Crystals	Urates.	None generally.	None.
Casts	Hyaline and granular; often fatty cells attached to casts.	All kinds; granular and hyaline, especially.	Granular, hyaline, and blood casts (?).
Morphological elements	Leucocytes, fatty epithelium.	Leucocytes, granular débris, fatty degenerated epithelium; red blood corpuscles sometimes.	Red blood corpuscles, large number of leucocytes.

Urine.—The following is a description of the urine ordinarily passed in chronic diffuse parenchymatous nephritis: Sp. gr. varies from 1015 to 1040. The urine is generally light yellow, cloudy, and foamy. Reaction is acid. The amount of albumin is generally large, 1 to 2, and even 5 per cent., being present (20 grammes or more being passed in the twenty-four hours). More is passed during the day than at night, and after exercise than after rest. The urea is diminished; especially does it decrease before death and uræmic attacks. The chlorates decrease with an increase of the disease. Nitrogen elimination is irregular. The sediment is generally abundant. Microscopical examination shows (*a*) granular and large and small hyaline casts, sometimes epithelial and fatty casts; (*b*) granular and fatty débris and fatty globules; (*c*) leucocytes, often enlarged and having undergone fatty degeneration; (*d*) red blood corpuscles are only found when there is an acute exacerbation of the chronic form; (*e*) often fatty degenerated epithelial cells. It requires a long time for certain mediums, such as quinine, iodine, potassium, and morphine, to be eliminated from kidneys affected with this form of nephritis.

Dropsy.—Next to the urine, this is one of the most important symptoms. It affects the skin, especially that of the lower extremities and the scrotum, and also the serous cavities. Sometimes it remains local. The dropsy is especially extensive where the nephritis is a complication of malaria or heart disease. It commences as a puffy, œdematous condition of the eyelids and in incipient cases is parallel with the diuresis. The œdema is caused by the hypo-albuminous condition of the blood and the retention of water. Dropsy is a constant symptom of this form of nephritis, occurring in 19 out of 20 cases in the writer's experience. This symptom is obstinate, and is the one for which the patient usually consults the physician. Interesting experiments made by Biernak have proven that the greater the dropsy the greater the amount of albumin in the urine, and the greater the interference with the excretion of urine the less will be the free hydrochloric acid in the gastric juice.

Gastric Disturbances.—Gastro-intestinal symptoms are common. The patient's appetite is generally decreased, the tongue is coated, thirst is increased, and there is often distress in the stomach. Frequently there is dyspepsia, nausea, vomiting, especially toward the end of the disease or when for any reason there has been a temporary suspension of the excretion of urine.

The causes of the gastric irritation are due not only to functional changes in the stomach, but to anatomical changes and to those of chronic gastritis. Sometimes the dyspeptic complications are purely nervous, and very often they are of uræmic origin. The ammoniacal odor of the patient is due to the decomposition of the uric acid in the digestive organs. The diagnosis is often made on this odor alone. The diarrhœal and dysenteric complications are due to the uræmic changes in the blood. Ulceration of the colon is often seen. The activity of the stomach glands is always decreased, and the amount of hydrochloric acid secreted diminished.

Blood.—The amount of red blood corpuscles and the percentage of hæmoglobin in the blood is greatly diminished, the former often falling

as low as 800,000 to each c.mm. of blood. The specific gravity of the blood is diminished and the white corpuscles somewhat increased. The urea in the blood is always increased. There is a tendency to hemorrhages.

Changes in the Heart and Pulse.—The heart is hypertrophied and dilated; especially is this seen with the secondary cirrhotic kidney. The longer the kidney affection has existed, the more surely may hypertrophy and dilatation be expected; and these symptoms are seldom if ever seen unaccompanied by slight dilatation of the left ventricle. The second aortic sound is generally accentuated, and an anæmic cystolic murmur heard over the base of the heart.

The writer examined 14 cases of chronic diffuse parenchymatous nephritis in which the diagnosis was made after a careful microscopical examination of the kidneys. In 2 cases the heart cavities were dilated without any valvular or arterial changes being found. In 12 cases the valves were thickened and the cavities dilated, although in most instances the changes were slight, and the heart could hardly be said to be the cause of the condition.

The writer believes that the cardiac dilatation, and not the hypertrophy, is the prominent clinical symptom of this form of nephritis, as far as the cardiac vascular changes are concerned. The pulse is changed; there is an increased tension, and later some stiffening of the arteries.

Nervous Phenomena.—These symptoms are less common with this form of nephritis than with any other. Symptoms due to the action of urea and the other waste products of the body upon the nervous system are seldom seen, unless during an acute exacerbation or near the close of the case, for the reason that the accumulation goes on so gradually that the system becomes habituated to its presence. When present they are marked by constant nausea, vomiting, headaches, convulsions, coma, stertorous breathing, or Cheyne-Stokes' respiration—in fact, all the classical symptoms of uræmia. It is well for the physician not to attribute certain symptoms, such as nausea, vomiting, and headaches, always to uræmia until he is sure that there is no other possible cause. Fleischer has proven that many of the symptoms are due to cerebral anæmia non-uræmic in origin.

Retinitis.—Often a patient will consult his physician for some trouble with his eyes, not dreaming that there is anything the matter with his kidneys. He will complain of indistinctness of vision, specks, or a mist before the eyes. The ophthalmoscope reveals retinitis apoplectica, fatty degeneration of the retina, or neuritis optica. These conditions are often developed to an extreme degree, not only when the disease runs a very chronic course, but even when it is more acute. The majority of the cases that have retinitis have hypertrophy of the left ventricle. Wagner found retinitis in 10 cases out of 157; Leroche, 8 cases in 100; and Rosenstein, 21 cases in 118.

Nutrition.—The evidences of malnutrition in this form of nephritis are always prominent. Loss of muscular energy and mental vigor are early indications of a rapidly advancing degeneration. Patients complain of always feeling tired and incapable of much physical exertion. The distressing symptoms come on, and the beginning of the end is

indicated when the malnutrition begins to affect the heart walls; then the whole picture of the case changes and the patient becomes rapidly worse.

Complications.—Inflammatory complications, such as pleurisy, pneumonia, pericarditis, and meningitis, are especially liable to occur during the course of this form of nephritis. Erysipelas, gangrene of the skin, and œdema of the lungs or epiglottis are also of frequent occurrence. Often the inflammations are subacute in their development. Chronic bronchitis is an almost invariable accompaniment of parenchymatous nephritis, and exists as a chronic catarrh with abundant expectoration. The more important complications are those which affect the heart and arteries; these are the result of malnutrition, assisted in many cases by the primary cause of the renal disturbance. When the cardiac hypertrophy, which is so constant, is associated with changes in the arteries, then we have a complication of grave importance which may lead to rupture, apoplexy, and sudden death.

Œdema of the lungs is the cause of more than one third of the deaths. Uræmia with complicating inflammation of the serous membranes stands next.

The following cases will show the frequency of complications as given by different writers: In 292 cases Frerichs found pneumonia 27 times, pleurisy 35, peritonitis 33, and pericarditis 13. Rosenstein in 114 cases found pneumonia 20 times, pleurisy 19, peritonitis 10, pericarditis 8. Oscar Meier found in 321 cases that there was an affection of the pleura, pericardium, and peritoneum, together, in 3 per cent. of all the cases; that there was an affection of the pleura and peritoneum in 15 per cent., and an affection of the pleura and pericardium in 7 per cent. The pleura alone was affected in more than 55 per cent., the pericardium in 16 per cent., and the peritoneum in 31 per cent.

Prognosis.—The structural changes which have occurred in the kidney after parenchymatous nephritis has been established do not admit of repair. Although cases of recovery have been reported, yet the writer does not think such a thing is possible.[1] He has seen a number of apparent recoveries. Under favorable conditions the extension of the disease may be delayed often for years, for a comparatively large number of tubes are unaffected in the earlier stages of the disease. Such kidneys stand as weakened organs especially subject to invasion by acute processes. In these cases a slight exposure or indulgence in alcohol may start again the process, and acute uræmia quickly terminate the case.

The liability of complications occurring at any time should lead always to a guarded prognosis. Of the varieties, the "large white kidney" is the most unfavorable, next the hemorrhagic form, and lastly the "smooth cirrhotic kidney." Persons suffering from this latter form often live a number of years, but statistics show that two to three years is the average duration.

The etiology does not affect the prognosis unless it has its origin in scarlatina or malaria. In gouty and rheumatic subjects the disease generally runs the longest course.

[1] The reported cases of recovery are in children, but the writer has found that if these cases are followed up there will be found to be a "lighting up" of the nephritis within four years after the "recovery."

In giving a prognosis the following points should be considered:

1. The degree and tenacity of the dropsy;
2. Quantity and quality of the urine passed in twenty-four hours;
3. Condition of the heart and general circulation;
4. Presence of uræmic symptoms;
5. The presence of secondary inflammation.

Clinical Picture of a Typical Case.—The following is a typical clinical picture of most cases of this form of nephritis: If the general health of the patient and his resisting power are maintained at the best standard, gradually the diuresis increases, the color of the urine becomes lighter, the specific gravity decreases, the quantity of albumin lessens, the cellular elements diminish, the dropsy decreases, the tension of the pulse increases, the second aortic sound increases, the heart beat becomes more powerful, the apex is carried toward the left, the patient gains strength, and the anæmia disappears. In all respects the patient is better, except for occasional headaches which remind him of the disease. With care this favorable condition may last for a long time, even years, until from some indiscretion an acute inflammation is set up in the already crippled kidney; then the life of the patient may be ended by some complication, as pneumonia or uræmia. Yet even when the case appears hopeless a second remission occurs with comparatively good health, to be followed again by an acute exacerbation. For years the patient may have these ups and downs. The changes in the dropsy and the apparent recoveries are the characteristic symptoms of this kind of kidney.

Diagnosis.—The following are the principal features which will aid one in deciding with which variety of kidney a patient is affected who is suffering from chronic diffuse parenchymatous nephritis.

The history of the case is by far the most important differential point. A satisfactory history is not always possible to obtain, especially as to the beginning of the disease; then the following considerations may be of help:

(1) **Large White Kidney.**—Œdema; extensive and tenacious anæmia marked.

Heart shows no hypertrophy.

Urine, highest specific gravity; largest amount of albumin; microscopical elements show fatty changes.

(2) **Chronic Hemorrhagic Kidney.**—Œdema, excessive and obstinate.

Anæmia, marked.

Heart, slight hypertrophy.

Urine, albumin abundant; red blood corpuscles in large amount and constant.

(3) **Smooth Cirrhotic Kidney.**—Œdema varies, often nearly disappears.

Anæmia, not marked.

Heart, hypertrophied; often dilated.

Urine, albumin varies; specific gravity lower; casts often disappear.

Chronic Diffuse Parenchymatous Nephritis and Amyloid Kidney.—The differentiation here is often difficult, for the etiology may be the same in both. The enlarged liver and spleen, with the recurrent

attacks of diarrhœa, are the important points to be considered, and are found in the amyloid kidney.

Chronic Diffuse Parenchymatous Nephritis and Chronic Diffuse Interstitial Nephritis.—A careful consideration of the etiology of the case is an important aid. Cardiac hypertrophy and vascular changes are much more marked in interstitial nephritis. A careful examination of repeated specimens of the urine will generally enable the physician to make the differential diagnosis. In interstitial nephritis the urine is larger in amount, of lighter color, and of low specific gravity; the albumin is scanty, and at times disappears; also there are comparatively few cellular elements. This kind of urine is directly opposite to that of chronic diffuse parenchymatous nephritis.

TREATMENT.—The two most important objects to be obtained by any plan of treatment are—

First, to arrest, if possible, the degenerative changes which are taking place in the kidneys, and restore healthy nutrition to the organ and the tissues generally.

Second, to prevent fresh invasion of still healthy renal tissue by careful regulation of diet, manner of life, and protection from changes of temperature. The first object can generally be accomplished by stimulating the functional activity of those portions of the kidneys which are still healthy. To one who has examined microscopically a large number of kidneys of this form of nephritis the folly of attempting to restore the inflamed and destroyed renal tissue is apparent, but by remembering that in the majority of cases the diseased tissue at first occurs only in isolated areas, between which are perfectly normal tubes and tufts, then the importance of preserving intact as long as possible these healthy portions is apparent. Of first importance is protecting the patient from change of temperature and chilling the surface of the body.

Clothing.—The clothing should be such that it will not interfere with insensible perspiration, but should keep out the cold. Flannels should be worn during the entire year; they can be light in summer, but still should always be woollen garments; a light flannel shirt should be worn at night.

Climate.—When possible the patient should live in an uniformly warm, dry climate, free from malaria, and where it is most comfortable to remain in the open air for a greater portion of the day. The sleeping apartments should be well ventilated, but the patient should be protected from draughts of air.

Skin.—The condition of the skin should be especially watched, and its function maintained by frequent tepid baths, followed by friction and rubbing of fats into the skin. Hot air baths should only be employed when the urine is diminished suddenly in quantity or uræmic symptoms are pronounced.

Diet.—The diet is most important. The food should be easily digested and nutritious; in this way only can the general nutritive processes be carried to their utmost—a desideratum so important. The writer believes that milk is the best all-around article of diet, but only in a very few cases should it be employed to the exclusion of all other articles of food. Often when subacute gastritis exists it is necessary to

predigest the milk, and in this way four or more quarts can be consumed in the twenty-four hours. Koumyss and buttermilk can often be substituted with advantage; milk with Vichy water is especially palatable.

The following are the conditions for dictating a restricted milk diet: (*a*) Acute exacerbation with rapidly diminishing urine; (*b*) uræmic symptoms; (*c*) a high tension pulse, for it has been proved that the pulse is lowered by a milk diet, while with a weak heart there is but little effect.

The writer is aware that a number of observers believe that an absolutely restricted milk diet is the great essential in the treatment of this form of nephritis, but his observations have warranted a more liberal plan of treatment. It must be remembered that each case differs not only as to the variety of nephritis, but as to the constitution of the patient, the anæmia, the condition of the digestive organs, the diuresis, and the degree of dropsy. Patients usually dislike milk after they have taken it exclusively for a long time, and it has been noted that after a time it loses its diuretic effect and causes a fall in the amount of solids and urea secreted. A mixed diet, the writer believes, is the best.

Heyden at his clinic has proven that in many chronic cases meat does not increase, but rather diminishes, the amount of albumin.

The following articles may be taken with benefit, using milk as the drink by choice: Eggs (soft boiled); fish; light meats, such as small quantities of veal, the white meat of chicken, etc.; vegetables and farinaceous food; bread, fresh cheese, pastry. Those who suffer from a slight dyspepsia or who have a failing heart are benefited by a moderate amount of stimulants, of which the light wines are to be preferred. Beer is specially injurious. Upon the least menace there should be no hesitation in returning to a simple milk diet—the exclusive food of the acute and subacute stages of the disease.

Exercise.—If the patient is able he should take moderate exercise, but never to the point of fatigue, and always with precaution against cold. Walking and riding are the best forms of exercise. Bicycling should be forbidden. When exercise is not possible massage should be resorted to.

For women marriage should be forbidden; at least pregnancy. Men should practise the greatest sobriety in sexual indulgence. Observations prove that sexual intercourse increases the symptoms. Tobacco should be used, if at all, very moderately or only when the patient is apparently well. Stimulation, except as before indicated, should be absolutely forbidden.

Medicinal Treatment.—The medicinal treatment pertains mainly to the most prominent symptoms, for, as before said, the most important consideration is the manner of life, diet, and clothing of the patient.

Our knowledge of the remedies which stimulate the functional activity of the kidneys is largely empirical. Most of the diuretics promote the flow of the watery elements of the urine rather than favor the excretion of the solid portions. The following drugs will give the best results in this form of nephritis:

Digitalis.—The amount of urine passed, the quantity of albumin, the character of the casts, and the extent of the œdema will determine the

extent to which it should be employed. When the urine shows concomitant acute inflammation, then it should be increased. If the urine is diminished during its use, it should be discontinued. The best preparation of digitalis to use for its action is the infusion made from the fresh leaves (this is important). The acetate or bitartrate of potash can be added to the infusion to increase its action. The writer has seen a number of times apparently hopeless cases of this form of nephritis—even those with extreme anasarca—rapidly improve on Fothergill's pills:

℞. Pulveris digitalis foliorum,
Pulveris scillæ radicis,
Hydrargyri chloridi mitis, *āā.* gr. xxiv.
M. et divide in pilulas numero xxiv.
Sig. One pill every four hours.

Care should be taken that the patient does not become salivated; this can often be prevented by using a solution of bicarbonate of soda as a gargle at frequent intervals.

Mineral Waters.—The alkaline waters are more useful here than in acute nephritis. Lithia water should be drunk freely. There is no question but that the epithelial accumulation in the convoluted tubes are eliminated more easily by the use of the alkaline waters.

Mercurials.—Formerly mercurials were used extensively in the treatment of this form of nephritis, and their use was persisted in over a long period of time. This plan of treatment has not proved satisfactory, and is now generally abandoned, only being used when the symptoms of an acute exacerbation occur, such as diminishing amount of urine with increasing œdema and dropsy. The best results are obtained with small doses, say $\frac{1}{10}$ grain, of calomel every half hour until a grain is taken, and this continued for a few days. Mercurials should not be given for any length of time.

Tonics.—While food is the most important help we have in carrying the nutritive processes to their highest point, we have two remedial agents of great value which we can use always with advantage—iron and cod-liver oil. The former is best given as the tincture of the chloride; both must be stopped if at any time the condition of the stomach contraindicates their use. They can be continued for a long period of time.

Diuretin has yielded in my hands but imperfect results. There is no question but that it increases the flow of urine in the majority of cases, but there are very few patients suffering from this form of nephritis that can stand its use for any length of time. Their stomachs rebel. The writer has obtained the best results with this drug in those cases in which the nephritis is complicated by cardiac insufficiency and weakness. Its diuretic action seems to be only temporary. The drug is used extensively in Germany in this form of nephritis. Jaborandi or the hydrochlorate of pilocarpine may be cautiously used in very urgent cases: either drug is prompt and efficacious, but requires great care in its use.

Stimulants.—Patients who suffer from dyspeptic symptoms are bene-

fited by a moderate amount of stimulants with their meals. Light wines are preferable. Whiskey, well diluted, can be given. Ale and beer should never be taken. Stimulants independent of food should never be allowed.

Hot Air Bath.—It is a cardinal rule in the treatment of this form of nephritis that no depleting measures should be applied unless in case of great emergency. Only when the other means at our disposal have failed to improve the action of the kidneys, and the dropsy is becoming excessive or the uræmic symptoms are developing rapidly, should the hot air bath, hydragogue cathartics, and powerful diuretics be employed. A weak heart or excessive dyspnœa is a contraindication for the use of the bath. The use of the bath should not be continued after the urgent symptoms have been relieved. Many cases are benefited by a hot pack once a day or once every other day, continued for a long time.

Chronic Diffuse Interstitial Nephritis.

Synonyms.—Chronic interstitial nephritis; Cirrhosis of the kidney; Gouty kidney; Contracted kidney; Granular kidney; Small red kidney; Granular atrophy of the kidney; Chronic desquamative nephritis; Renal sclerosis.

This form of nephritis is characterized clinically by its slow and insidious development, and anatomically by an atrophy of the secreting parenchyma secondary to the development of new connective tissue. This fibrous outgrowth is the essential characteristic of the disease, and ends in more or less complete destruction of all the anatomical elements of the organ.

Two varieties of kidneys are found in this form of nephritis, distinct as to their cause and in their microscopical appearance:

1. **Genuine inflammatory cirrhotic kidney**, an independent primary affection, developing like cirrhosis of the liver.

2. **Degenerative cirrhotic kidney**, a sequence to arterio-sclerosis. In these kidneys no doubt the essential lesion is in the secreting cells first, and the connective tissue outgrowth is secondary. The kidneys of the second class are much more common than those of the first.

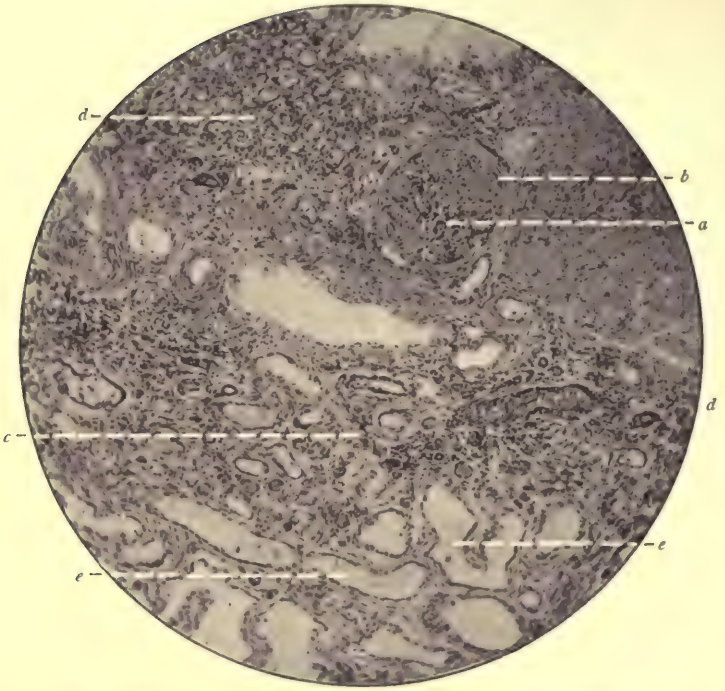
Many observers add a third variety of kidneys—namely, those which started as large white kidneys or smooth cirrhotic kidneys, and in time became contracted into the kidneys of chronic diffuse interstitial nephritis. The writer's experience in post-mortem examinations does not bear this out, and he believes all the kidneys of true cirrhosis can be embraced under these two heads. The gross appearances of these two forms of kidneys will be described together. Only on microscopical examination can a diagnosis be made.

Pathological Anatomy.—Both kidneys are reduced in size and both are involved to the same extent.

Gross Appearances.—The kidneys often weigh not more than an ounce each. The capsule is thickened and adherent; the surface is irregular and granular; it presents a puckered appearance. The kidneys are either hyperæmic and red or anæmic and of a pale gray color; the gray is the more common. The red is the more developed form, is smaller than the gray, the granulations on its surface are larger.

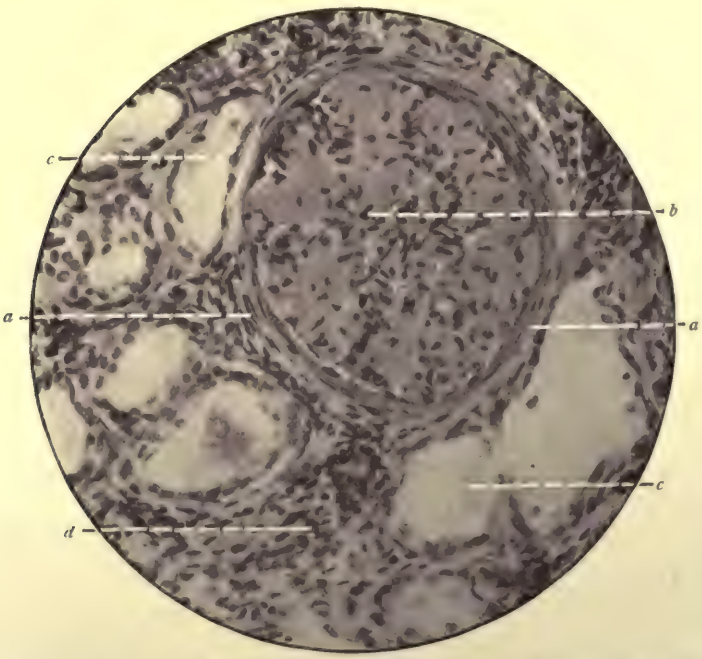
PLATE XIII.

FIG. 1.



Chronic Diffuse Interstitial Nephritis. *a*, Atrophied Glomerulus undergoing Hyaline Change; *b*, New Connective-tissue around Glomerulus; *c*, New Connective-tissue between Tubules; *d*, New Connective-tissue Area; *e*, Dilated and Denuded Tubules. x 80.

FIG. 2.



Chronic Diffuse Interstitial Nephritis. *a*, Increase of Connective-tissue about Glomerulus; *b*, Hyaline Degeneration of Vessels of Glomerulus; *c*, Tubules almost entirely Denuded of Epithelium; *d*, New Connective-tissue. x 210.

Photographed by Henry S. Stearns, M. D.

Small cysts containing a clear, transparent liquid are frequently seen on the surface.

On section the cortex is thinned, especially at the site of the cicatrices. It is reduced to one third its normal size, and has the same color as the surface. The pyramids are shortened and redder than normal, the pelvis is wider, and there is much fat around the organ. The small arteries often stand out prominently. Deposits of urate of soda, which appear as white lines through the medullary portion, are often seen. These deposits are invariably seen when the renal disease is associated with gout (Plate XIII.).

Microscopical Examination.—Under the microscope the granular elevations are seen to correspond to the portions of the cortex which remain—the sunken portions to the atrophied parts which have been replaced by connective tissue. The following are the changes seen in the different elements of the kidney:

(1) *Connective Tissue.*—The increase of connective tissue is widely distributed throughout the organ, especially in the cortex between the medullary rays, and more especially about the veins. The first step of the induration change is a round cell infiltration in the intertubular tissue and about the tufts. This tissue becomes fibrillated, contracts, and destroys both tubes and tufts. Areas of small round cells are often seen on the edges of the old fibrous patches, showing that an acute process is going on and the new connective tissue is forming. The capsules of the glomeruli in the affected areas are thickened and compressed by the concentric rays of new connective tissue which surrounds them. When the new tissue is diffuse the changes in the tufts are most marked. When the process apparently starts around the vessels it is much less. Between the pyramids the increase of connective tissue is seen, but is much less in amount than in the cortex and more diffuse. (See Plate XIII. Fig. 1.)

(2) *Glomeruli.*—When there is much thickening of the capsule of Bowman and narrowing of the vas afferens the glomeruli begin to atrophy, the epithelium in the capillaries is loosened, and that covering the tufts is desquamated. The tuft then undergoes hyaline degeneration, and is converted into a granular nucleated structure which does not allow the blood or artificial injections to pass through it. There is but slight change, if any, in the capsular epithelium. Some tufts, especially in the areas free from connective tissue, appear normal, but on careful examination it will be found that the cells of these tufts are increased and that the capsule of Bowman is thickened. In this form of nephritis there are changes in practically all the tufts. (See Plate XIII. Fig. 2.)

The changed tufts excrete albuminous urine, which not only packs together the degenerated and desquamated epithelium, but flows down the tubes, carrying the granular débris with it.

There is apparently some connective tissue between the capillary loops, the great increase of nuclei seen being derived either from (*a*) the desquamated epithelium covering the tufts, (*b*) the endothelium of the capillaries, (*c*) the new connective tissue between the loops, or (*d*) the white corpuscles within or without the vessels.

(3) *Tubes.*—The epithelium of the uriniferous tubules undergoes the same changes as those described under Parenchymatous Nephritis.

The cells of the tubes in the connective tissue areas are atrophied or replaced by cuboidal cells. Some of the tubes are entirely denuded of epithelium; others are dilated and the cells have undergone granular fatty changes. Many tubes are greatly dilated, which is due to plugs of granular débris forming in the portions constricted by the new connective tissue and the pressure of the urine dilating the tubes above the plugged portion. The degeneration of the renal epithelium is much less widespread than in parenchymatous nephritis. Large areas of normal tubes are seen in kidneys in an advanced stage of the disease. This condition no doubt accounts for the slowness in development in the disease and the prolonged life of patients suffering from this form of nephritis. Many tubes are collapsed and almost obliterated by the connective tissue. Many of the tubes contain casts and granular débris, but a much smaller number than is seen in parenchymatous nephritis.

(4) *Vessels* (Plate XIV. Fig. 1).—The arteries show an advanced sclerosis. The tunica adventitia is thickened; the inner coat shows an inflammatory growth (endarteritis obliterans), which in places almost obliterates the lumen of the vessel. The media is also thickened by an increase of connective tissue which has replaced many of the muscular fibres. The capillaries become obstructed, and finally obliterated. In the second form of kidney—the degenerative cirrhotic kidney—the changes described above, occurring in the small and medium sized arteries, are characteristic and prominent. They are enough to establish the diagnosis and classify the kidney. By these changes in the small arteries and in the vessels of the glomeruli the circulation through the kidneys is impeded. This leads to degeneration of the renal epithelium, which no longer receives enough blood to nourish it. It atrophies and new connective tissue develops in its place. This is the sequence of the changes in these kidneys, the arterial being the primary change.

In the first form (genuine cirrhotic kidney) the primary and characteristic change is in the glomeruli, the tubular and connective changes being secondary.

Heart.—Associated with the cirrhotic kidney changes in the heart are *always* found. The most prominent is cardiac hypertrophy, and this is always found if the heart be carefully examined, although in many cases seen at autopsy the secondary dilatation is so pronounced that the hypertrophy may be overlooked. Sometimes the hypertrophy is so excessive as to produce the cor bovinum. Many theories have been advanced to account for this hypertrophy. Before stating the one which the writer believes to be the correct one it may not be amiss to briefly give the various theories:

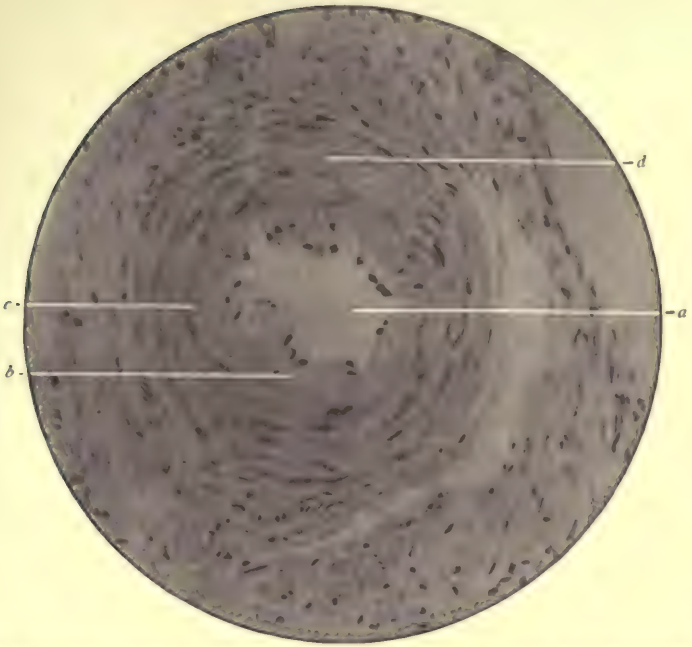
(*a*) *Mechanical, from Blood Pressure.*—Explains the hypertrophy by changes in the larger renal capillaries and increase in the fluid elements of the blood—all causing increase of cardiac work (Traube).

(*b*) *Arterio-capillary Fibrosis.*—The thickening of the outer and inner coats of the renal arteries, with atrophy of the muscularis, gives a constant obstruction in the renal circulation which must be overcome by hypertrophy of the heart (Gull and Sutton).

(*c*) *Chemical Changes.*—The urinary excretions, being retained in the general arterial system, cause an irritation resulting in hypertrophy.

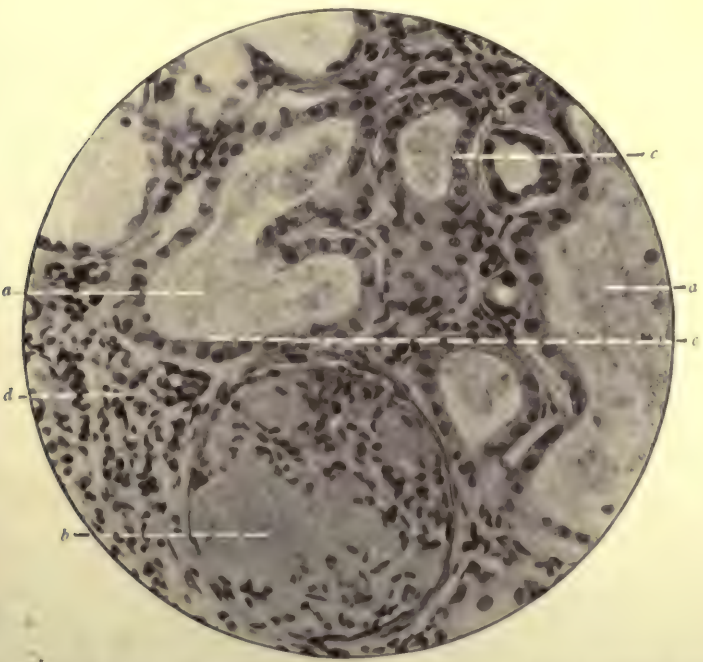
PLATE XIV.

FIG. 1.



Endarteritis Obliterans, from a Cirrhotic Kidney. *a*, Lumen of Artery; *b*, New growth Obliterating Lumen; *c*, Fenestrated Membrane; *d*, Tunica Media. x 210.

FIG. 2.



Amyloid Degeneration of the Kidney. *a*, Lumen of Tubules Filled with Granular Material; *b*, Amyloid Material in Glomerulus; *c*, Degenerated Renal Epithelium; *d*, New Connective-tissue. x 210.

Photographed by Henry S. Stearns, M. D.

A gradual accumulation of material in the blood for a long time adds to the work of the heart.

The most probable explanation of the hypertrophy, to the mind of the writer, and the one also most generally accepted, is that originally proposed by Cohnheim. He believes (1) that the circulation through the kidney, which is regulated by the contraction or dilatation of the small renal arteries through their muscular coat, depends solely upon the excrementitious matter in the blood requiring urinary excretion. To accomplish this object without disturbance of the general circulation requires increased heart action and general arterial contraction. This explains the hypertrophy seen in parenchymatous nephritis as well as in interstitial nephritis. (2) He also believes that when parts of both kidneys have been destroyed by new connective tissue, to maintain the blood pressure in the obstructed but still normal areas an increased force of the heart is necessary, with general arterial contraction.

General Arterial System.—The explanation of the *cause* of the general arterial tension has been given. It must be remembered that it is the dilated and failing, and not the hypertrophied, heart which is to be feared in nephritis. In the writer's opinion dilatation is due to changes in the muscular fibres of the heart, especially those of the left ventricle, brought about, first, by the blood, loaded with the uræmic poisons which have not been eliminated by the kidneys, failing to properly nourish the myocardium; second, by the coronary arteries obstructed by fibroid changes and furnishing an insufficient quantity of blood to the heart. This leads to general arterial muscular hypertrophy. A hypertrophied heart is constantly pounding against tense arteries. What is the result? Degeneration of the arteries takes place; permanent fibroid disease is established—a far more serious condition than can ever result from sympathetic arterial contraction.

Associated with this form of nephritis is found a general arteriostenosis change, not only in the middle but also in the external and internal coats. Atheromatous and calcareous changes are generally found along the aorta, especially with the degenerative cirrhotic kidney.

Etiology.—This form of nephritis occurs most frequently in males and between the ages of forty and sixty. It is seen to develop in this country almost exclusively in two classes of cases: (1) In those with an inherited or acquired lithæmic or gouty condition, whose urine almost always contains an excess of uric acid—persons who "live well," exercise little, and stimulate moderately; (2) those whose arteries tend to degenerate early, in whom the fibroid diathesis is well marked—a condition most commonly inherited and accompanied by defect in development of the nutritive processes and often by hepatic functional weakness.

While the prolonged use of alcohol, especially the stronger liquors, often induces the cirrhotic kidney, the writer believes that excessive eating, especially of meat and rich foods, is more liable to develop this condition than excessive drinking. It is a clinical fact that active brain-workers, who exercise little, live well, and drink moderately, are especially liable to develop this form of nephritis through arterial sclerosis. Alcohol and hard mental work cannot go together without

injury to the kidneys. Gout is not as important an etiological factor in this country as in England. Charcot has proved that gout may exist for a number of years and the kidneys not be affected. The cirrhotic kidney is not an attendant upon acute gout; it is seen most frequently in those who live all their lives just upon the border line of an acute attack. The cirrhotic kidney may be caused by chronic lead-poisoning, yet the writer has examined after death a number of cases of chronic lead-poisoning without finding the cirrhotic kidney, and believes it stands in a much less causal relation than is generally supposed. Koster found the cirrhotic kidney in only 2 out of 37 cases examined; from 10 to 40 per cent. is the proportion given by various observers.

The rheumatic poison is less active in producing the cirrhotic kidney than the gouty; still, this kidney has developed after severe cases of acute articular rheumatism, and is sometimes seen with chronic rheumatism; but as these cases often suffer from chronic endocarditis (valvular heart disease), it is difficult to decide which is the true cause.

Syphilis produces the cirrhotic kidney by first inducing a specific disease of the renal arteries.

Diseases of the genito-urinary passages, such as stricture of the urethra, pyelitis, may cause interstitial nephritis.

The senile kidney so commonly found at the post-mortem examination of old people is, as far as the gross and microscopical appearances are concerned, a cirrhotic kidney, and is due primarily to changes in the renal bloodvessels, just as atheroma is commonly found in the large arteries of old people. It is not a true nephritis, but only a part of the "normal" changes of old age.

Symptoms.—The development of this form of nephritis often passes unrecognized. Many cases are discovered only on the post-mortem table. So long as a sufficient amount of normal renal tissue remains for the removal of waste products from the body, the only symptom may be an increase in the amount of urine passed, the patient being obliged to rise a number of times at night to urinate.

Sooner or later the results of the defective elimination will manifest themselves. General muscular weakness and an unaccountable lassitude will gradually develop; the patient feels that he is growing old, and is unable to apply himself with accustomed energy to work. Possibly at this time some slight œdema will be noticed about the ankles, most marked at night and disappearing in the morning. There is gradual loss of appetite, and marked dyspeptic symptoms appear early, accompanied by acidity and anorexia, most marked in the morning.

Preceding or following these gastric symptoms various evidences of the action of the poison upon the nerve centres, such as pressure on the head, headache in the occipital region, and pain in the back of the neck, will appear and resist all ordinary treatment: this is especially seen in the case of nervous persons. Irritability of temper, fretfulness, treacherous memory, and sleeplessness are all symptoms marking the development of this form of nephritis.

We will now consider certain symptoms which are always present when the disease is fully developed:

Urine.—The quantity of urine is greatly increased, so as often to lead to suspicion of diabetes, 3000 to 6000 c.c. being passed in twenty-

four hours. It is clear and often of a slightly greenish color, and there is little or no sediment. The specific gravity is low, varying from 1006 to 1012. The urine is acid. Albumin is generally present, but in small quantities, 0.5 per cent. being the maximum amount. At times albumin may be absent for a few days or a portion of a day, so that repeated examinations of the urine may be necessary in certain suspected cases with urine of low specific gravity. In the beginning a normal amount of urea is eliminated, and may even reach 500 grains per day, but later it is diminished. Casts are never present in large quantities, and when found are usually of the small hyaline variety. A few lymph cells are generally found, but no blood corpuscles unless an acute attack of nephritis develops.

Heart.—One of the most characteristic evidences of this form of nephritis is found in the condition of the heart. The heart is hypertrophied, especially the left ventricle. Bright records that in 100 cases there were 52 with hypertrophy, 34 of which were without valvular lesion and 11 with aortic disease. In 22 per cent. there was no other ground for the hypertrophy than the nephritis.

In 12 cases with cirrhotic kidneys examined on the autopsy table the writer found 10 with diseased hearts (hypertrophied and dilated), apparently secondary to the kidney lesions.

Increased arterial tension with its firm pulse, ringing second sound, and forcible apex beat may give warning years in advance of the developing hypertrophy and foretell the form of nephritis to be expected. When hypertrophy is fully developed the apex beat will be displaced to the left and will be more forcible than normal.

Cardiac dulness is increased to the right and left, the second aortic sound will be accentuated on account of the resistance of the blood pressure in the aorta and the arterial sclerosis. The first sound is weak on account of the arterial changes and the slight dilatation of the ascending aorta. Frequently a systolic murmur is heard at the apex, depending upon coincident endocarditis or the beginning of dilatation of the ventricle.

As long as the hypertrophy keeps up the arterial tension, and so regulates the urinary secretions, the condition of the patient is favorable, but sooner or later, unless death follows from accidental complications early in the disease, degenerative changes will occur in the heart muscle and dilatation with cardiac insufficiency appears. Then the pulse loses its tension, becomes smaller, more frequent, and a little irregular. Slight physical exercise now affects the patient, and he becomes short of breath and is troubled with palpitation. Symptoms referable to the lungs may now appear, such as those of mild or persistent bronchitis or congestion of the lower lobes, or even spots of lobular pneumonia.

As a further evidence of imperfect heart action œdema appears, first of the lower extremities, and later this may become more general.

It is needless to discuss the various opinions which have been advanced by different writers to explain the almost constant evidence of cardiac hypertrophy with this form of nephritis. Briefly they are as follows: Buhl thinks the primary change is in the heart muscle itself—namely, a myocarditis. Cohnheim says local resistance in the kidney increases the blood pressure and so causes the hypertrophy. Johnson

found excessive thickening of the middle coat of the arterioles resulting from tonic contractions. Gull and Sutton found thickening of the outer and inner coats with atrophy of the muscularis. Another theory is that the urinary secretions, being retained in the blood, cause an irritation resulting in the hypertrophy. This theory has lately been strengthened by the works of Israel.

Traube's theory gives two reasons for the increase in the heart work: first, more water taken in and less excreted for a long time, and, second, the increase in peripheral resistance from the deranged circulation in the kidneys.

Appearance of Patient.—The face is generally pale with a slight yellow hue. There is a weary, listless expression; the eyelids are slightly swollen; the temporal arteries are tortuous; the skin of the body is dingy, dry, and scaly, and there is but little tendency to sweat, and stripes remain where the skin is scratched. The patient is anæmic. An examination made by the writer of the blood of 10 patients suffering from this kind of nephritis showed an average reduction of the hæmoglobin from 13 to 8, the red blood corpuscles from 5,000,000 to 3,000,000, and the amount of blood serum increased.

Œdema.—The œdema is very variable and may be entirely wanting. For one or two weeks it will be present about the ankles every night or after long standing or walking; this may be followed by a still longer period when it is entirely absent. When œdema occurs to any extent it must be regarded as due to a secondary parenchymatous inflammation.

Gastric Disturbances.—Dyspeptic symptoms are frequently the earliest to attract the patient's attention. He complains of loss of appetite and anorexia. Later nausea and vomiting, with profuse diarrhœa, often utterly uncontrollable, may develop.

Eyes.—An ophthalmoscopical examination of the eye will reveal in the retina yellowish white spots with small inflammatory exudates and often minute hemorrhages. The retinitis is important in diagnosing this form of nephritis.

Brain.—The brain symptoms which may develop during the course of a cirrhotic kidney are the following: (1) Disturbance of motor centre: uræmic convulsions, epileptic convulsions, tremors, localized contraction of muscles, especially those of neck and face.

(2) Disturbance of psychical centre: delirium, hallucinations, vertigo, coma, melancholia.

(3) Sensory centre: deafness, blindness, and hemiopia.

(4) Centre of respiration: dyspnœa, Cheyne-Stokes respiration, laryngeal spasms.

(5) Centre for heat regulation: hypo- and hyperthermia.

Even when fully developed the cirrhotic kidney may run a comparatively mild course for years, until finally, under the strain of accidental disease or complication or from gradual exhaustion, the patient suddenly develops uræmic symptoms and passes into a state of coma from which he never rallies.

The danger which constantly menaces the person suffering from this form of nephritis is the involvement of the comparatively normal portions of the kidney in an acute inflammatory process from exposure,

overwork, or indiscretions. In such cases the urine will suddenly decrease in quantity, the albumin will increase, and marked œdema develop before the uræmic seizure closes the scene. In studying the cirrhotic kidney one is impressed with the fact that clinically very many cases will divide themselves into groups according as the prominent symptoms are confined to a single organ. These cases can be grouped under the following heads:

(1) *Cardiac Cases.*—Here the symptoms are mainly cardiac, and apparently the renal cirrhosis, the arterial degeneration, and the resultant cardiac changes all develop about the same time. This class of cases is generally found among persons suffering from gouty manifestation and with a marked fibroid diathesis. They show at autopsy the arterio-sclerotic kidneys, and include at least 20 per cent. of all the cases of renal cirrhosis.

(2) *Cerebral Cases.*—These are the cases that suffer from persistent and intense headaches, attacks of vertigo, numbness, formication, and even temporary paresis of a single limb.

(3) *Gastro-intestinal Cases.*—This group of persons will suffer almost solely from symptoms pertaining to the gastro-intestinal canal, persistent nausea and vomiting, attacks of profuse diarrhœa uncontrolled by any remedy.

(4) *Cases showing Progressive Weakness.*—This class often presents symptoms which should call attention to the kidneys. The patients, however, are many times treated throughout the whole course of their illness for nervous prostration, anæmia, or senile decay. Gradual advancing weakness and exhaustion are the only evidences of kidney changes. This group of cases is much more common than is generally supposed.

Complications.—Many of the conditions found in this form of nephritis are to be looked upon as part of the disease rather than as complications.

Cerebral hemorrhage is by far the most frequent complication, for the cerebral arteries are early affected with the fibroid changes occurring throughout the general arterial system; capillary aneurysms are formed; these rupture from some unusual strain, such as cerebral hyperæmia or excess in cardiac action. This is the form of kidney disease most frequently associated with cerebral apoplexy.

Inflammations of serous membranes are common—peritonitis, pericarditis, and sero-fibrinous pleurisy especially so.

Chronic bronchitis with acute exacerbations is common. Often the bronchial inflammation leads to a catarrhal pneumonia.

Lobar pneumonia is a complication of grave import.

Neuro-retinitis or retinal hemorrhages are seen in many cases. Also hemorrhages from mucous and serous surfaces and into the substance of organs are liable to occur. Œdema glottidis sometimes occurs. To show the frequency of the complications, in 100 cases of this form of nephritis reported, 17 died of cardiac disease, 15 of apoplexy, 18 of pulmonary disease (11 of severe bronchitis and emphysema, 7 of pleurisy and pneumonia).

Prognosis.—When once the pathological changes which characterize this form of nephritis are established, the tendency is for them to ad-

vance. Fibrous tissue, when once developed, is never removed, but slowly extends until by its contraction the organ affected is destroyed.

The prognosis is generally bad, but the patient may live for a number of years, most of the time enjoying the pursuits of an active life. I know a gentleman, still alive, in whom the diagnosis of interstitial nephritis was made fourteen years ago. While this is an exceptional length of time for a fatal termination to be delayed, still eight and ten years are not unusual. The average duration of this form of nephritis is from three to five years.

There are no distressing symptoms as long as the heart compensates, but as soon as the heart begins to fail weakness, dyspnœa, and dropsy will appear, which are readily amenable to treatment at first, but later increase in spite of the greatest care. The severe œdemas which are sometimes seen late in this form of nephritis indicate a failing heart rather than a superimposed parenchymatous inflammation of the kidneys. It is a grave symptom. A careful watch should be kept on the pulse as indicating a developing uræmic or cardiac condition.

A constant source of danger which menaces the patient is uræmic intoxication, which generally appears late in the disease and is often fatal.

Even with the severest symptoms, such as Cheyne-Stokes respiration, persistent vomiting, and a water-logged condition, the patient may live for weeks or even months.

Treatment.—Although we cannot hope to remove the new connective tissue in the kidney when this disease is once established, and although we know that further fibroid development will surely take place in spite of all we can do, still, by removing the exciting causes and freeing the blood as far as possible from irritating substances, we can delay the fatal termination for years.

General Hygiene.—*Clothing.*—As this variety of kidney lesion is especially susceptible to the reflex effects of chilling the surface of the body, the patient should be always warmly clad and flannels should be worn next the skin both day and night. This will prevent the sudden and acute inflammations of the organ which are so fatal.

Climate.—When possible the patient should reside in a warm, dry climate, subject to but slight variations of temperature. A continued residence in one place is not desirable. The beneficial effects of change of air, food, and surroundings always act as physical and mental stimulants. The patient shows marked improvement from such changes. In this country the cold winter months can be passed in Southern California, on the Gulf coast, at Thomasville; the spring months at Atlanta, Asheville, or Virginia; and the summer in the north, Saratoga having an especially favorable climate.

Skin.—It is imporant that the skin should be kept in as favorable condition as possible. For this reason tepid baths taken before retiring are of advantage. These can be given daily or only two or three times a week. By taking the baths at bedtime there is less danger of exposure and of taking cold. They must be given with care. The effect of the baths will be enhanced by friction of the skin. In some cases massage, followed by oil inunctions, will be of benefit. The importance of keeping the skin in good condition can readily be appreciated when we

remember that by it is removed nearly as much of the waste products each day as by the kidneys.

Exercise.—The amount and kind of exercise will depend upon the stage of the disease and the condition of the patient. The most important guide as to the latter is the condition of the heart. In the early stages, especially when there is marked hepatic derangement, horseback riding should be prescribed, but it should never be carried to the point of fatigue. An out-of-door life is important. In those cases with cardiac involvement, and especially with the evidences of cardiac failure, the mildest form of exercise, such as walking short distances or riding, should be ordered. The effect of exercise on the heart should be noted before absolute rules can be made. A long sea voyage is beneficial to this class of cases.

Food.—A restricted milk or skim-milk diet is not beneficial in this form of nephritis. Although recommended by some, it will be found by experience that it is not possible to confine a patient for months to this diet. Milk when well borne is a valuable adjunct to a general diet. What may be called an anti-gout diet, containing only a moderate amount of albuminoid food and little, if any, of the starches, is the best, as is also a mixed diet, consisting of light, nourishing, easily digested, and non-irritating food.

An exclusive milk diet may, however, be employed when an acute exacerbation threatens the patient. Light wines, well diluted, may be used with meals. Alcohol and tobacco should be prohibited. Above all things, constipation must be avoided, and this can often be controlled by a suitable diet.

Alkaline waters, so highly recommended by European observers, are especially useful. The natural lithia waters give much better results than those made by dissolving lithia tablets in water.

Medicinal Treatment.—The writer does not believe that it is possible to act on the connective tissue already formed in the kidneys, either by mercury or the iodide of potassium, the use of which is recommended by some.

Diuretics.—Particularly those cases of interstitial nephritis which are associated with cirrhosis of the liver are greatly benefited by small doses of the bichloride of mercury given for protracted periods, and in cases where the urine is of high specific gravity and loaded with uric acid a dose of "gray powder" and quinine at bedtime for a few nights, and followed by a saline in the morning, will show marked beneficial results. The continued use of mercury is not permissible in rheumatic (?) cases or cases of lead-poisoning, and when given should not be continued for any long period of time.

The diuretic action of digitalis will not be required unless some acute inflammation of the kidney supervenes.

Iodide of potassium is one of the most valuable remedies in the treatment of interstitial nephritis. As a diuretic, as a substance to increase glandular activity, and as a remedy in arterial sclerosis it is invaluable. Its best effect is seen in cardiac disturbances depending on arterial sclerosis. It should be employed for several weeks at a time.

Heart.—In acute heart failure alcohol (champagne, cognac), strong coffee, and camphor will give the best results.

In subacute or chronic cardiac weakness the cardiac tonics are useful. Of these digitalis stands first. When used in combination with strophanthus and nitro-glycerin its cardiac tonic action is obtained without an increase in arterial tension. To maintain the nutrition of a hypertrophied heart and delay dilatation as long as possible a general tonic treatment is essential. For cardiac palpitation cold compresses over the chest, bromide of potash, nitro-glycerin, or tincture of valerian may be given.

Tonics.—Although persons suffering from this form of nephritis are generally anæmic, it is found that iron will agree with but few. Indeed, in many cases the nervous symptoms are aggravated by its use. Where iron can be borne it will have a special beneficial effect in improving the nutrition of the heart. Cod-liver oil and the hypophosphites are useful in a large proportion of cases.

Gastric Disturbances.—In treating the gastric and intestinal disturbances it should be remembered that diarrhœa is often a means of excreting the urea, and by suddenly stopping it uræmia may result. Gastric and intestinal derangement may be treated with hydrochloric acid, bitters, alkalies, etc. When vomiting is persistent, it will often be necessary to put the patient on koumyss or peptonized milk for a few days.

Dropsy.—Although dropsy is less frequent in this than in any other form of nephritis, still general anasarca may develop in the later stages. If cardiac stimulants fail, a resort to mechanical interference may be necessary. Aspiration should not be delayed too long. It not only relieves the general symptoms, but often gives marked relief to the kidneys. Punctures of the skin of the extremities or scrotum are often necessary.

Uræmia.—This is best treated by increasing the secretion of the skin, intestines, and kidneys or by overcoming the cardiac weakness. The treatment does not differ from that used when the same condition occurs in other forms of nephritis, except that bleeding in some of these cases gives wonderful results. For cerebral congestion cold compresses to the head or leeches to the back of the neck are useful.

Asthma.—This is a distressing symptom. Morphine should be used preferably hypodermically. Throughout the disease, when any anodyne is to be used, opium is always to be preferred. Nitro-glycerin and amyl nitrite generally give marked relief.

AMYLOID DEGENERATION OF THE KIDNEYS.

Amyloid degeneration of the kidneys is a chronic disease in which both organs are affected, and is considered by some an event in the process of a chronic nephritis. It is frequently associated with chronic diffuse parenchymatous nephritis, especially with the large white kidney; also sometimes, but much less commonly, with chronic diffuse interstitial nephritis, and again it may exist as the only renal lesion. With some it is an open question as to whether the amyloid degenera-

tion of the vessels is a complication of an inflammatory process or is the cause of the retrograde change. Amyloid degeneration rarely occurs in the kidneys only, but is usually associated with similar changes in the spleen, liver, intestinal mucous membrane, and glands. It is attended by albuminuria, and in the later stages by dropsy. Hence older writers considered it a form of Bright's disease that occurred in individuals who were cachectic. As this change is not an inflammation of the kidney, it cannot be considered a nephritis.

As early as 1842, Rokitansky described the pathology of amyloid degeneration. Virchow, when he first examined the nature of the amyloid material, discovered that when treated with a solution of iodine it assumed a dark brownish or mahogany color, and therefore thought the amyloid material was similar to vegetable cellulose. Friedreich and Kekulé proved that it was a nitrogenous substance, more fitly classed as an albuminate. It is insoluble in gastric juice at the body temperature. In addition to the mahogany color which the amyloid material assumes on the addition of iodine solution, it becomes a darker brown, blue, violet, or greenish color when dilute sulphuric acid is added to the section stained with the iodine solution. Upon the addition of anilin-violet or methylanilin the amyloid material is stained a bright ruby red and the healthy tissue blue or indigo color. These color changes are really a chemical reaction.

It is generally believed that amyloid degeneration is a circulatory process which takes place in the organ affected, and begins in the muscularis of the arteries, and may extend until the whole artery is involved or may remain limited to the muscular coat. It may not only affect all the coats of the artery, but may appear in patches, forming circumscribed areas which extend partially or wholly around the vessels. The epithelial lining of the arteries remains intact. Experiments to prove the integrity of the vessels have been made by Litten. He injected an amyloid kidney with a lime solution colored with Berlin blue. The injection was made into the kidney under the pressure of a column of mercury. The vessels could be traced in the longitudinally cut section and the spots of amyloid material readily seen. In none of these did the injected fluid extravasate, which showed that the vessel wall was intact and that hemorrhage in the amyloid must be uncommon.

The most extensive amyloid changes are found in the glomeruli and the vasa afferentia. The amyloid material is not absorbed after being deposited, and consequently acts as a foreign body, interfering with the circulation. This impairment of the circulation interferes with the nutrition of the epithelial elements, and hence these usually become fatty, may break down, and only the nuclei remain visible. The tubules of the kidney usually appear enlarged and contain granular and fatty material, but they may be empty and collapsed. Their epithelium may remain unaffected, though generally it has undergone fatty degeneration. (See Plate XIV.)

PATHOLOGICAL ANATOMY.—The changes in amyloid degeneration of the kidneys may be so slight as to pass unrecognized at the autopsy table, and be discovered only upon the application of a chemical test or upon microscopical examination. This slight change may take place

within the vessels without the parenchyma of the organ or the interstitial tissue being affected. However, there is usually parenchymatous and interstitial change, so that from a clinical standpoint it may be looked upon as a diffuse nephritis, the question remaining whether the nephritis or the amyloid process began first.

All kidneys affected with amyloid degeneration may be included under the following three heads:

(1) Amyloid changes in the bloodvessels without changes in the epithelium and interstitial tissue of the organ. This is a rare condition, but is sometimes seen.

(2) Amyloid degeneration of the kidneys, with chronic diffuse parenchymatous nephritis (the large white kidney)—a common condition.

(3) Cirrhotic kidney with amyloid changes.

(1) **Pure Amyloid Kidney.**—This kidney is of rare occurrence, but still is sometimes seen. It can develop in one month. There are no particular symptoms with this form except slight urinary changes and the enlarged spleen. The writer recalls a case in which there was amyloid degeneration of the vasa recta without glomerular affection. The case was not diagnosed during life, as there was no albuminuria.

As a rule, the kidney is smooth and glistening, unchanged or slightly increased in size and weight, pale and anæmic, yellowish white in color; its consistency is softer than normal and the capsule is easily removed.

On section the cut surface may look quite normal, or, if there is any thickening, it is confined to the cortex, which is pale and anæmic like the surface. The changes in the kidney may be so slight, however, that they may pass unrecognized unless tested with iodine solution or examined under the microscope. The pyramids have a bluish red color, the striæ are obscured, and the Malpighian corpuscles glisten with the translucent, homogeneous amyloid material. Upon the application of Lugol's solution there will appear reddish brown spots which are the affected glomeruli.

On *microscopical examination* the amyloid material is readily detected. The walls of the vessels appear thickened and permeated with a glassy, homogeneous material, which often has the appearance of being in flakes. The amyloid material is found most frequently and in the greatest abundance in the capillary loops of the Malpighian body. It is also seen in the afferent and efferent arteries. Still, cases are seen in which these are unchanged and only the vasa recta are involved. It may be seen in the capillaries of the interstitial tissue, the vasa recta, and occasionally in the capillaries of the medullary portion and the membrana propria of the tubules. The parenchyma and interstitial tissue appear normal.

(2) **Amyloid Kidney with Chronic Diffuse Parenchymatous Nephritis.**—Amyloid degeneration of the kidney is frequently found in connection with parenchymatous changes in the kidney, and occurs oftenest in the large white kidney. It is seen especially in connection with phthisis. In this kidney also the amyloid change may not be observed until tested with iodine solution. The kidney is large, heavy, sometimes twice the normal weight—is grayish white, and frequently yellow spots are visible. The capsule is easily removed and the surface beneath is smooth and glistening. On section the cortex is seen to be

thickened, and the glomeruli appear as specks which turn brown on the application of iodine solution. (See Plate XIV. Fig. 2.)

Microscopic Examination.—Amyloid changes are seen in the Malpighian tufts, in the arterioles, and often in the wall of the tubes. Never are the cells infiltrated with amyloid material, but they are broken down, swollen, granular, and fatty. A glomerulitis is seen in some cases. The tubules of Bellini appear enlarged, thickened, and filled with granular or fatty cells.

(3) **Cirrhotic Kidney with Amyloid Changes.**—Amyloid degeneration occurs less frequently in the cirrhotic kidney than in the preceding form. When it does occur, amyloid changes can be observed both in the parenchyma and interstitial elements of the organ. This kidney often complicates gout or syphilis. The kidney is smaller and tougher than normal, generally reddish brown, but sometimes pale or grayish white in color, with a thickened, adherent capsule which tears away part of the substance of the kidney when it is removed. The surface of the kidney is rough, and presents indentations and elevations which are the result of the increase and contraction of the connective tissue in the cirrhotic process or the result of the atrophy of the organ in those portions where the amyloid change is the most extensive.

On section, the cortex presents about the same color as the surface. Small granules may be seen scattered through it, and sometimes small cysts filled with a clear fluid. By some the latter are considered characteristic of the amyloid change.

Microscopically, the connective tissue increase is seen and may be extensive. The arteries contain amyloid material, and often the lumen of the vessels is diminished if not rendered impermeable. More or less degeneration of the parenchyma will always be seen, some of the tubes being completely denuded. Thrombosis of the renal veins is often observed in the amyloid kidney. Sometimes it is seen to have entirely closed the vessel. Apparently, this has not led to any changes in the renal tissue, so it is fair to suppose that a collateral circulation was established.

In some cases a microscopical examination of the cardiac muscle shows a myocarditis as a result of an amyloid change in the small vessels in the heart wall.

ETIOLOGY.—Amyloid degeneration is not a primary lesion, but a change occurring in the course of some preceding pathological condition. It takes place not only in the kidneys, but usually affects the spleen and the liver at the same time. When only one of these organs is affected it is the spleen. The mucous membrane of the intestines, the lymphatic glands, the thyroid gland, the suprarenal capsules, and the heart may also be affected.[1] It has not been possible to produce the disease by means of experiments, and its nature has not been fully determined. Amyloid degeneration occurs mainly in cachectic individuals, and, while it is without much question derived from the blood and is considered by many an infiltration, yet it probably does not exist in the blood as amyloid material, but is derived from the fibrin factors. Some authors suggest that it is formed in some particular place in the

[1] In 200 cases of amyloid disease the spleen was affected in 98, the kidneys in 97, the liver in 63, and the mucous membrane of the intestines in 65.

body as a necrotic area, and carried thence and deposited in the organ affected; some suggest that it is formed direct from the tissue elements in which it is found; while others think there is a preceding pathological change in the tissues which causes the proper conditions for the deposit of the amyloid material.

Amyloid degeneration may occur at any period in life, but is most frequent between the ages of twenty and thirty, diminishing in frequency before and after that age. It occurs in both sexes with nearly the same frequency—somewhat oftener in males than in females. Of 144 cases showing amyloid degeneration of the kidneys reported by Fehr, the following are the ages given:

1 to 10 6	31 to 40 36	51 to 60 7
11 to 20 24	41 to 50 23	61 to 70 7
21 to 30 43		

The writer's autopsy records show that out of 76 cases of amyloid kidneys, 45 were in males and 31 in females.

Cases occur in which there can be found no antecedent disease, but they are nearly always preceded by some pathological condition to which the disease can be traced. There is nearly always a history of some chronic ulcerative process, especially in cases marked by pus formation. The diseases with which it most frequently occurs are chronic pulmonary tuberculosis, syphilis, long standing ulcerations of bones or skin, and ulcers of the intestinal tract.

Of 43 cases, 17 were suffering from advanced pulmonary tuberculosis. Over half of the cases had cavities and were complicated by tubercular ulceration of the intestines. Litten reports that 70 per cent. of the cases had tuberculosis of the lungs, and 31 per cent. of these had ulcerative processes in the intestines.

It also occurs in cases of continued inflammation, in empyema, bronchiectasis, pyelo-nephritis, psoas abscess, chronic ulcerative peritonitis, carcinoma, lupus, and quite frequently in chronic ulcers of the legs. A few cases have been reported in which gout, rheumatism, lead-poisoning, and leucæmia have been accompanied or followed by amyloid degeneration of the kidneys, of the spleen, liver, or intestinal mucous membrane.

Symptoms.—As amyloid degeneration of the kidney is preceded by some chronic process, the development of the symptoms must necessarily be obscure. The more pronounced symptoms of the causal disease are likely to mask in part or entirely the obscurely developing symptoms that appear with the amyloid change. It should also be remembered that the symptoms vary greatly in character, the variation depending largely upon the extent and degree of any diffuse nephritis which may be present. It is not uncommon, therefore, for the amyloid degeneration to be overlooked and not be recognized until the autopsy reveals its presence. The development of a similar change in the spleen, liver, or intestines, or in all these organs, simultaneously with the change in the kidneys, is apt to add confusion to the condition and cause variation in the symptoms.

The symptoms of amyloid degeneration are not pronounced in their development. The patient usually notices first that he is getting weaker,

is short of breath when climbing stairs or exercising, is becoming very pale, and that his complexion has a waxy appearance. He observes that he passes more urine than formerly, that he has to micturate oftener, and that frequently he has to get up during the night to pass water. The abdomen may appear full, and sometimes there is a sensation as if something in the upper portion were pulling down. An enlarged spleen or liver may cause a disagreeable sensation when lying down on either side, and, on account of difficulty in breathing, causes the patient to sit propped in bed. This dyspnœa may be due to the anæmic condition of the blood or to the pressure from the enlarged spleen or liver, or to both. The nature of the urine as a direct indication of the disease depends much upon whether the parenchyma or the interstitial tissue is affected most. It varies with the extent of the change in each.

The Urine.—The urine is greatly increased in quantity, and the patient may pass over a hundred ounces in a day. It is light in color, sometimes straw colored, and sometimes looking like water. It is faintly acid, of low specific gravity, ranging from 1005 to 1015, and contains but little sediment. In the scanty sediment there can usually be found large hyaline, small coarsely granular, and broad waxy casts, to which adhere fatty globules. Some of these casts may respond to the iodine test, but very seldom is this the case. Tubular epithelium which sometimes has undergone fatty change, and very rarely red blood corpuscles, are seen in the sediment. Leucocytes, fatty epithelium, and fatty débris are often seen. Albumin is present in varying quantities—in the majority of cases from 0.4–0.8 per cent. In a few cases it may be entirely absent. When albumin is not present it may be presumed that the amyloid change has affected the vasa recta and that the glomeruli have escaped uninjured. When there is an accompanying nephritis, albumin is present without exception, and generally in large quantities. Senator and Edelfsen distinguished between the urine of nephritis and that of amyloid kidney by the presence of a large amount of globulin in the latter. The amount of urea excreted is usually less than normal, but the low vitality of the patient more than the presence of the amyloid change may be the cause of this. It varied from 1.28 per cent. to 1.07 per cent. when the urine was averaging 2.460 c.cm. per day and sp. gr. 1010 in a case under the writer's care. When the amyloid change is associated with the large white kidney, albumin is always present, and usually in large quantities; the amount of urine is diminished, the specific gravity normal or increased, and the color dark. When it is associated with the small cirrhotic kidney the quantity of urine is always large, and only toward the end of the disease or when there is severe diarrhœa or fever is it diminished; but even then the specific gravity remains low. With this form of amyloid kidney the albumin is constantly secreted, but in diminished quantities; the urea is also increased.

Dropsy.—There is usually more or less dropsy, but it is not extensive and is not a constant symptom. In 114 cases it was found 82 times. General anasarca is more common, with an effusion into the subcutaneous tissue or serous cavities. There may be some œdema of the feet, especially at night, and fluid may accumulate in the abdominal

cavity. When œdema occurs it is likely to remain persistent. The anasarca is largely influenced by the preceding disease and the influence of that disease upon the blood. Wagner reports that it occurs in one third of the cases of amyloid kidney following phthisis, in one fourth after rheumatism, and in one half after syphilis.

Gastric Disturbances.—These are not infrequent in amyloid degeneration of the kidneys. Nausea, loss of appetite, severe and often dangerous vomiting, and diarrhœa are very common. These indicate that in all probability there is an accompanying amyloid degeneration of the vessels and mucous membrane of the intestines. The stools may be bloody.

Heart.—In the pure amyloid degeneration of the kidneys the heart is not hypertrophied and there is no cardiac lesion resulting from the renal change, but in the amyloid cirrhotic kidney the heart often presents the same condition as in the simple cirrhotic kidney—namely, hypertrophy of the left ventricle. Some authors think that when the hypertrophy does occur it indicates that the cirrhosis preceded the amyloid, and that when the amyloid change precedes the cirrhosis the heart is normal in size or smaller than normal.

In cases of cirrhosis of the kidneys with amyloid degeneration retinitis and uræmia may develop, but these conditions do not occur in cases of simple amyloid degeneration.

The temperature remains normal. The respiration may be disturbed, either by the pressure of an accompanying enlargement of the spleen or liver or both, or by the anæmic condition of the blood. Dyspnœa may be so severe as to prevent the patient from lying down, but does not become stertorous, as in the dyspnœa of nephritis.

Diagnosis.—In considering the diagnosis of amyloid degeneration of the kidney it should be remembered that it is a condition occurring with or following some other pathological lesion—a causative and coexisting disease. The presence, then, of some disease, as phthisis, tertiary syphilis, chronic ulcers, disease of the bones, etc., antecedent to the kidney lesion arouses the suspicion of amyloid degeneration. An enlarged spleen or liver may aid in differentiating between amyloid kidney and chronic nephritis.

If there be no albuminuria, the condition of amyloid change will not in all probability be recognized. The abundant pale, clear urine with low specific gravity, containing little sediment, few or no red blood corpuscles, and with albumin in varying quantities, aids in the recognition of the disease. There is generally some dropsy, not as extensive as in the large white kidney, but more than occurs with the cirrhotic kidney. Ascites is often present on account of the associated changes in the liver and spleen, and a persistent diarrhœa may aid the observer in recognizing the presence of extensive amyloid degeneration involving the mucous membrane of the intestines. In 76 cases of amyloid kidney, 48 cases had the change in liver, spleen, and kidney; in 20 cases the spleen and kidneys alone were involved, in 4 cases the liver and kidney, and in 5 cases the kidney alone. The clinical fact remains that the liver and spleen are not always enlarged proportionately to the amyloid change of the kidney. Cardiac hypertrophy is absent and uræmic symptoms are very rare. The cachectic condition of the patient, the pale

waxy skin, and the increasing weakness, all suggest amyloid degeneration in the kidneys. While the disease may occur at any period in life, it is usually found between the ages of twenty and thirty, and is insidious in its onset.

When amyloid degeneration occurs in the large white kidney or the small cirrhotic kidney, the symptoms produced by the condition preceding the amyloid may so mask those of the latter that it will remain unobserved, but even in this the cachexia, the condition of the skin, and the character of the urine will aid in recognizing the condition. The absence of increase in arterial tension or hypertrophy of the heart points to the amyloid kidney.

The following is a synopsis of the various symptoms presented by the different forms of amyloid degeneration of the kidney:

	Pure Amyloid Kidney.	Amyloid Large White Kidney.	Amyloid Cirrhotic Kidney.
Etiology.	*Syphilis, Bone Diseases.*	*Phthisis, Bone Disease.*	*Gout, Rheumatism.*
Urine:			
Amount	Increased.	Normal or diminished.	Normal or increased.
Color	Pale yellow, clear.	Reddish, cloudy.	Pale yellow.
Specific gravity	Low (1006).	High.	Low.
Urea	Normal.	Diminished.	Increased.
Albumin	Little (1 per cent.) or absent.	Constant, 2-4 per cent.	Constant, little.
Casts	Few, hyaline.	All forms.	Few.
Sediment	Absent.	Abundant epithelium, fatty débris.	Slight.
Cardiac hypertrophy	Absent.	Not constant, slight.	Present.
Dropsy	Moderate.	Excessive.	Slight.

An enlarged spleen and liver, dropsical effusions, and diarrhœa are complications seen in all forms.

Prognosis.—The prognosis in amyloid degeneration of the kidney is usually unfavorable, though it may be years before the kidney reaches the stage of atrophy. The duration of the disease is very variable, and, like the prognosis, is much influenced by the nature of the preceding disease and the general condition of the patient. If the causal disease can be arrested or cured, the amyloid process may be stayed in its progress and the patient's life prolonged for a number of years. The duration of the disease is shortest in chronic phthisis, longer in bone disease, and longest in syphilis. It is not at all probable that a retrogressive process takes place in vessels once affected with amyloid degeneration, but, as in other diseases of the kidneys, portions of the organs may be unaffected, and may be able to carry on the function of the organ for a long period. We know, however, that the presence of amyloid material in the kidney adds gravity to the coexistent disease, and indicates the presence of a marasmus, and possibly similar changes in other organs which will probably prove fatal to life in the end.

The fact that albumin has disappeared from the urine is no indication that the disease has been cured or that the condition of the patient is necessarily better.

Treatment.—When the kidneys have undergone amyloid degeneration it is impossible to administer anything which will cause the disappearance of such degeneration, so that in one sense it may be looked upon as an incurable disease. There have been a few cases reported as cured, but it is doubtful if more than the arrest of the process was

attained. The most to be hoped for at the present time is to arrest the disease and build up the strength and general health of the patient.

The main question is prophylaxis. When the physician has recognized a condition which portends amyloid degeneration, he should take every possible means to remove the condition. With the limited means at our disposal it seems impossible to stay the process in many cases of chronic ulcerative pulmonary tuberculosis, but where there is a constant drain upon the system, as by necrotic areas of bone, it is necessary that these areas be removed even if it requires the amputation of a limb. In addition to combating the causative disease by remedies suited to the particular case, much can be done in the way of prophylaxis by building up the general condition of the body. If there be a history of chronic syphilitic affection, mercurials and potassium iodide should be employed with the hope of removing the cause. Some clinicians advocate the use of the iodides whether there is any definite history of syphilis or not, claiming that a great number of cases are due to that cause whether the history bears out the conclusion or not, and also that the iodides exercise a beneficial result upon the bloodvessels when there is amyloid affection. These drugs should not be carried to their physiological effects.

Table giving the Etiology, Condition of the Urine, and Prominent Symptoms of the Principal Diseases of the Kidney.

		Acute Congestion.	Passive Congestion.	Acute Diffuse Nephritis.	Chronic Diffuse Nephritis.		Amyloid Degeneration.
					Chronic diffuse parenchymatous nephritis.	Chronic diffuse interstitial nephritis.	
Etiology.		*Drugs (cantharides), Vaso-motor Paralysis.*	*Heart or Lung Diseases.*	*Toxic Poisons (scarlatina, cantharides).*	*Secondary Acute, Alcoholism, Exposure.*	*Gout, Fibroid Diathesis, Syphilis.*	*Suppuration, Syphilis.*
Condition of the urine.	Amount.	Scanty.	Scanty.	Diminished.	Increased.	Very abundant	Increased.
	Color.	Dark.	Dark red.	High colored.	Pale.	Pale.	Pale, clear.
	Sp. gr.	1030.	1028.	1030.	1010–1014.	1004–1012.	1014 or 1005.
	Urea.	Normal.	Normal.	Reduced ½.	Diminished.	Diminished.	Normal.
	Albumin.	Large amount.	Moderate amount.	Large amount.	Abundant.	Scanty, sometimes absent.	Moderate amount.
	Casts, sediment.	Hyaline blood corpuscles.	Hyaline red blood corpuscles.	Hyaline and epithelial casts.	Hyaline, granular, or fatty casts, white or red blood corpuscles.	Large hyaline, finely granular.	Hyaline, granular, waxy.(?)
Dropsy.		None.	Depends on primary disease: stationary; lower extremities.	Severe (seldom absent).	Always present; often excessive.	Not constant; slight.	Considerable.
Nervous phenomena.		None.	None.	Prominent.	Not marked.	Very prominent.	Absent.
Cardiac hypertrophy.		None.	Depends on primary disease.	None.	Present.	Present.	Absent.
Gastric disturbances.		Present.	Generally present.	Prominent.	Chronic dyspepsia.	Slight.	Absent.
Causes of death.		Non-fatal.	Heart failure, infarctions.	Uræmia, œdema of lungs, inflammation of viscera.	Uræmia, cardiac insufficiency, inflammation internal organs.	Cerebral hemorrhage, uræmia, cardiac failure.	Exhaustion.

In treating the amyloid degeneration when it has once fully developed there is not much to be done besides treating the causal condition

and securing if possible its removal or arrest. Having attended to the conditions producing the disease, the general nutrition of the patient should next receive attention. In order that the system may be built up the patient should be given the best of food, a mixed diet being preferable. The bowels should be regulated and moderate exercise enjoined. Tonics, arsenic, iron, and especially the iodide of iron and cod-liver oil, will prove beneficial. Proper baths for the purpose of keeping the skin in good condition should be employed. Having cared for the causing disease and the general nutrition of the patient, the remaining treatment consists in modifying, if possible, symptoms after they arise.

RENAL HYPERÆMIA.

Active Renal Hyperæmia (Active Congestion of the Kidneys).

Normal secretion of the kidneys is maintained by uniformity of the circulation, and any disturbance in this uniformity will result in the disturbance of the amount or character of the secretion. Any disturbance in the circulation that tends to cause degeneration of the normal epithelial elements will naturally lead to disturbance of function, which will, in all probability, manifest itself in the character of the urine.

It is generally believed that the watery elements of the urine are filtered off in the glomeruli, and that when the epithelium of the glomeruli becomes damaged it fails to restrain the albumin, and albuminuria is the consequence. In renal hyperæmia there is one of two main causes acting: (1) a disturbance in the vaso-motor control of the arterioles of the kidneys, resulting in hyperæmia, but not in albuminuria; or (2) the action of some irritant, as a poison, which causes a renal hyperæmia always accompanied by more or less albuminuria. It has been demonstrated that the renal epithelium is very susceptible to any influences producing a change in the normal relation of the kidneys, and that the action of any irritant or even compression upon the renal artery for only a few minutes may result in albuminuria. We can understand, then, why the hyperæmia of vaso-motor paralysis does not cause an albuminuria, for there is no irritant in the circulating blood, the epithelium has an abundant supply of oxygen, and the increase in pressure results only in polyuria.

Pathological Anatomy.—Active renal hyperæmia, apart from any other pathological condition, is so rarely the cause of death that but little opportunity is offered for the study of its pathological anatomy. In cases severe enough to result in death the cause of the hyperæmia is so severe as to produce an acute inflammatory process which might more properly be classed as an acute nephritis.

Both kidneys are affected at the same time, unless we consider those cases in which one kidney is affected with a compensatory hyperæmia

when its fellow is affected by some condition which interferes with its function.

Gross Appearances.—In active hyperæmia the kidneys are enlarged; the surface is smooth and darker in color than normal. The kidney is softer than normal, and the capsule is non-adherent. The hyperæmia does not always affect the whole organ, but may affect only certain areas, in which case the hyperæmia is most marked in the pyramids. In certain places ecchymotic spots may be seen.

On section the vessels are seen to be congested and a dark fluid follows the knife. The congested Malpighian bodies appear as dark red spots. The epithelium of the tubules generally remains normal, but when the irritation has been very severe there may be recognized a slight granulo-fatty change. The epithelium of the glomeruli is more often affected; it may be swollen and even desquamated. The interstitial tissue is usually infiltrated with serum, and if the process continues it assumes more of the character of an inflammatory exudation. The capillary vessels and the small arteries are seen to be engorged with blood, and hemorrhagic extravasations occur in places.

Etiology.—Active hyperæmia of the kidneys is, as stated above, due to one of two main causes: (1) A disturbance in the vaso-motor control of the arterioles of the kidneys, resulting in dilatation of these arterioles, and consequent hyperæmia. This hyperæmia from vaso-motor paralysis is likely to occur in cases suffering with Basedow's disease, diabetes insipidus, hysteria, or emotional disturbances.

(2) To the action of some irritant poison affecting the kidney and producing arterial congestion. The poison may be due to the entrance of some chemical agent into the circulation from without, or to the action of some poison produced within the body by bacterial life. The action of the poisons in cases of the acute infectious diseases often produces a more or less extensive inflammatory process, and hence these cases are better considered under the head of acute nephritis.

The most common among the drugs which produce active hyperæmia of the kidneys are cantharides, oil of mustard, turpentine, cardol, cubebs, copaiba, potassium chlorate, potassium nitrate, antimony tartrate, carbolic and sulphuric acids. Some drugs, as arsenic and phosphorus, produce renal hyperæmia, but are followed by more serious pathological changes than usually result from simple renal hyperæmia. Cantharides is the drug which causes the greatest number of cases of renal hyperæmia, and affects the kidneys in a similar manner whether taken internally or used as an external application. The same may be said of mustard, and the same effect is produced by turpentine whether given by inhalation or internal administration.

Symptoms.—The symptoms of renal hyperæmia vary according to whether the hyperæmia is caused by the presence of an irritant or by vaso-motor disturbance. In the latter case the main symptom is polyuria with a desire to pass water frequently. The urine is abundant, pale, clear, of low specific gravity, and non-albuminous. When the hyperæmia is due to some irritant, as cantharides, there is also a desire to pass the water often, but the urine is scanty and may be entirely absent. It is high colored, of high specific gravity, and contains more or less albumin. The albumin is generally abundant and coagulates sometimes

spontaneously, and may even form masses in the bladder and prevent micturition. The urine also contains some hyaline casts and red blood corpuscles.

Sometimes the patient complains of pain in the lumbar region over the kidney and sometimes in the glans penis. But in the majority of cases there is no pain present.

In cases where the renal hyperæmia is due to the action of an irritant, as cantharides, disturbances are present in other organs of the body. Especially is this true of the gastro-intestinal tract, when the mucous membrane of the stomach and intestines is in a state of congestion. This may be so severe as to produce accompanying symptoms of pain, nausea, and diarrhœa.

Diagnosis of renal hyperæmia is greatly aided by the history of the case. Where the hyperæmia is due to a vaso-motor disturbance in the kidneys, there is usually a history of nervous phenomena, with increase in the amount of urine, or of the presence of diabetes mellitus, hysteria, or perhaps of Basedow's disease. When the hyperæmia is due to some irritant, there is the history of the administration of a drug which could cause the condition. Thus, if a patient has been using cantharides internally or locally, or has been taking turpentine either by the stomach or by inhalations, and suddenly develops the symptoms of renal hyperæmia, the condition will offer no difficulty in its recognition.

The condition of the urine is of vast importance in recognizing the presence of active renal hyperæmia. In that form due to vaso-motor disturbances the presence of a great amount of non-albuminous urine of low specific gravity and pale color readily indicates the renal condition. When the hyperæmia is caused by an irritant, the scanty urine, the great amount of albumin, which has a tendency to coagulate, the dark color, and the high specific gravity are plain indications of the congested condition of the kidneys.

Prognosis.—The prognosis in active hyperæmia of the kidneys is good. Seldom is the renal hyperæmia alone the cause of death, and the congestion of the kidneys passes away upon the removal of the cause.

Treatment.—In considering the treatment of active renal hyperæmia it should be noted that prophylaxis is of great importance. In the administration of those drugs which have an irritant effect upon the kidneys the amount given should be so estimated as to produce no ill results, and the urine should be carefully watched for the first indication of renal irritation—namely, diminution in quantity, increase in specific gravity, increase in color, and the presence of albumin.

When the active hyperæmia exists the most important consideration is to determine the cause and remove it as speedily as possible. Upon the withdrawal of the irritant the symptoms generally begin to subside. The patient should be put to bed, kept in a room at a temperature of about 75° F., and treatment must be directed to the kidneys. Cupping over the kidneys in the lumbar region may give relief. Bloodletting may be resorted to in most serious cases, but usually is not necessary; besides, it has the undesirable effect of weakening the patient. The patient should be given plenty of diluent drinks and a drastic purge. The hot air bath or the warm vapor bath may be used with benefit in some cases. It is necessary that the action of the skin should be em-

ployed to aid in reducing the hyperæmia, and therefore it is important to produce free diaphoresis.

The use of drugs in active renal hyperæmia is not extensive. Besides those used to secure the action of the bowels and perhaps in the securing of diaphoresis, camphor is about the only one that can be recommended for this condition.

Passive Renal Hyperæmia (Passive Congestion of the Kidneys).

Passive renal hyperæmia is a more common and a more serious condition than active renal hyperæmia. It is a condition that does not occur as a primary disease, but depends upon some preceding interference in the local or general circulation. By interference in the renal circulation certain changes are produced within the kidneys varying in extent with the duration of the interference. The nature of the disturbance also causes a varying condition in the kidneys. When it is due to some disturbance within the kidney itself or in the renal vein, one kidney alone may be affected, but when it is due to some obstruction in the inferior vena cava or to some interference with the general circulation, as that which attends certain cardiac and pulmonary diseases, both kidneys will suffer alike. When the disturbance is in the general circulation there is not only the congested condition of the veins of the kidneys, but at the same time the arterial pressure is diminished, and this, added to the venous congestion, produces more rapid and extensive changes in the renal elements than occur when the congestion is due to some local cause.

Pathological Anatomy.—In passive renal hyperæmia both kidneys are affected alike, except in those rare cases when the interference in the circulation is unilateral. In these cases one kidney will undergo pathological changes, while the other remains normal or becomes slightly enlarged from a compensatory hypertrophy. In the usual form of passive renal congestion, where the disturbance is bilateral, the appearances presented are quite characteristic. The kidneys are usually somewhat enlarged, thicker, and more rounded than normal; dark in color and firm in consistency, often presenting a stony-hard consistency. Their weight is increased out of proportion to their size. The surface is smooth and the capsule non-adherent, except in chronic cases, when it may be nodular and somewhat adherent. The stars of Verheyen are prominent, full, and distended. When the congestion is of recent origin the veins and capillaries are uniformly dilated.

On section blood flows from the cut surface, the medullary portion of the kidney appears redder than the cortex, and sometimes there may be seen hemorrhages into its pelvis or into the ureter. The cortex is of a dark bluish red color streaked with lines of bright red, due to the congested bloodvessels. The Malpighian bodies are often dark and prominent. In them the most characteristic changes are found.

Microscopical Appearance.—Blood corpuscles and an albuminous fluid are often present in Bowman's capsule. The epithelium of the tubules is sometimes granular, fatty, and opaque, and each individual cell is often distinctly seen; the cells may contain some pigment de-

posited from the broken-down red corpuscles. The lumen of the tubules is larger than normal, and partially filled with coagulated albumin or detritus, and in rare cases red blood corpuscles are seen. The epithelium lining the tubes is often flattened. The veins of the entire organ are enlarged, tortuous, and varicose. The connective tissue is usually increased, especially when the congestion has been of long duration, producing "cyanotic induration." Malnutrition, resulting from the imperfect circulation in the organ, may prepare the way for more extensive anatomical changes, and, though the Malpighian corpuscles and urinary canals do not undergo extensive changes at first, yet with the malnutrition of the organ and the long continued congestion the epithelium of the tubules becomes quite granular, fatty, and finally breaks down. A few red blood corpuscles and an albuminous fluid are often present in Bowman's capsule; the capsule of Bowman is thickened as well as the membrana propria of the urinary tubules. The capillaries which make up the glomeruli are dilated, generally filled with blood, and their walls thickened. This I consider characteristic of this form of kidney. The connective tissue between the urinary tubules and between the vessels is increased, and the arteries, veins, and capillaries become thickened and round cell infiltration is visible.

ETIOLOGY.—Passive hyperæmia of the kidneys is dependent upon a disturbance in the circulation within the organ. This disturbance may be caused by interference with the general circulation or with the local circulation. The causes of the disturbance in the local circulation may be an interference in the kidney itself, in the renal vein, or in the inferior vena cava. Thus the presence of a neoplasm within the kidney, a thrombosis in the renal vein, or pressure upon that vein by a tumor in some adjoining organ may produce a disturbance in the local circulation. In these cases the passive congestion will be limited to one kidney, but this condition does not often occur, and usually both organs are affected. Any condition which interferes with the return circulation through the inferior vena cava will tend to produce a passive congestion in which both kidneys are involved. This interference may be produced by a phlebitis or thrombosis of the inferior vena cava, by pressure in some adjacent organ, as from the gravid uterus, by peritonitic adhesions, by ascites, and, some writers claim, by the rapidly enlarging liver when that organ is affected with amyloid degeneration.

By far the most common cause of passive congestion of the kidneys is a disturbance of the general circulation, and the most frequent cause of that disturbance is some cardiac or pulmonary lesion. Pulmonary lesions are less important than cardiac diseases as etiological factors. Chronic valvular disease is the condition above all others which produces passive congestion. Failure of compensation in mitral regurgitation and mitral stenosis is a most frequent cause of disturbance in the general circulation, for these usually result in tricuspid insufficiency, and when this takes places the blood is dammed back into the inferior vena cava and the whole circulation is disturbed.

In like manner, aortic insufficiency or stenosis may lead to a high grade of general congestion, which finally results in renal congestion. Any condition, therefore, which results in cardiac failure or dilatation

necessarily results in impairment of the general circulation, and finally produces congestion of the kidneys. Thus a myocarditis, fatty degeneration of the heart muscle, idiopathic hypertrophy and dilatation, all tend to the same end—namely, renal congestion. Chronic nephritis, which first produces hypertrophy, then dilatation, is a frequent cause of cardiac failure, which causes marked interference with the return circulation and congestion of the kidneys. In these cases the renal congestion is more serious on account of the pre-existing nephritis.

Diseases affecting the pericardium also frequently produce passive congestion of the kidneys by affecting the heart, disturbing its function, and thus interfering with the general circulation. Thus pericarditis with effusion or suppurative pericarditis may produce serious renal congestion. Pericardial adhesions may cause a similar effect. In like manner arterio-sclerosis, narrowing of the aorta and aneurysm, narrowing of the coronary arteries, or morbus Basedowii, may by their effect upon the heart cause a high grade of passive renal congestion.

There are certain conditions of the lungs which produce congestion of the kidneys; the disturbance, however, in these cases depends upon the interference with the general circulation. This interference is most frequent in those conditions which by reason of compression or obliteration of the pulmonary arterioles and capillaries interfere with the circulation through the lungs. The existence of chronic interstitial pneumonia or chronic emphysema is very likely to cause this disturbance. It may also occur in the later stages of asthma, in chronic tuberculosis of the lungs, in diseases of the pleura, especially in pleurisy with effusion, in empyema, or in tumors occurring in the lungs or in the mediastinum.

It should be remembered that any condition which interferes with the heart action may produce renal congestion, and therefore it may occur in cases of anæmia, in the acute infectious diseases, in the marasmus of old age, and in those who for any reason have become very weak.

Symptoms.—Passive hyperæmia of the kidneys is usually accompanied by other symptoms, due also to the general disturbance of the circulation, and may pass unrecognized for a considerable length of time. It is always difficult to decide how largely these symptoms are dependent on congestion of the kidneys. The patient will probably first notice that he has difficulty in breathing when taking severe exercise or going up stairs, and, in fact, finds he becomes short of breath very easily. Swelling of the feet and legs or ascites may first attract his attention and cause him to seek medical advice. Palpitation of the heart, cyanosis, dyspnœa, or œdema is frequently present, and causes the patient anxiety and often real distress. Along with the disturbed circulation there is often a cough, which, though due to congestion of the lungs, nevertheless contains a nervous element.

The alimentary tract is often disturbed, followed probably by loss of appetite, nausea, and vomiting. The patient loses flesh, becomes anæmic, dropsy increases, and general anasarca may result before death supervenes.

The history of these cases varies, and some patients, after being very ill, improve and become comparatively comfortable for a time. They

vary thus from bad to better, then worse again, thus having repeated attacks sometimes covering a long period. No matter how hopeless the case may seem to be, there is the possibility of a return to a fairly comfortable condition. Some cases, however, grow steadily worse, and end fatally without any marked intermission of the symptoms.

Symptoms which distinguish passive congestion of the kidneys are *limited almost entirely to changes in the urine*, and are distinct from other symptoms which accompany the disturbance in the general circulation at the time.

When there are but slight structural changes in the kidneys the urine may be about normal in quantity, the specific gravity is but little changed, and but little albumin may be present. There are usually present, however, a few small hyaline casts and a few epithelial cells. The urine is acid in reaction, and when allowed to stand for a time there is a deposit of urates. Accompanying the albumin there is often blood, the amount of which is usually small, but it may be found in considerable quantity. When the disease has been of longer duration and greater structural changes have taken place, the quantity of urine is diminished and the specific gravity is higher, reaching as high as 1030. The color is darker than normal. It contains a moderate amount of albumin, hyaline casts, and red blood corpuscles, without there being, however, a sufficient amount of blood to color the urine. The excretion of uric acid is greater than in health.

The diminished amount of urine is supposed by some to be due to the venous congestion and the consequent slowing of the circulation, especially through the glomeruli. The presence of albumin may be due to the increased blood pressure or to an impairment of the glomerular epithelium from the impaired arterial circulation, which causes faulty nutrition.

Diagnosis.—Passive renal hyperæmia is usually recognized by the general condition of the patient and from the history of the case. There is frequently present some valvular lesion, cardiac failure, pulmonary congestion, or venous stasis pointing to a passive congestion involving the kidneys. The condition of the urine is important as indicating the nature of the affection. The dark color, the high specific gravity, the presence of a moderate amount of albumin, a few hyaline casts, and a small amount of red blood corpuscles,—these conditions, in connection with the history and general condition, render the diagnosis a simple one.

In cases of renal embolism blood may appear in the urine, and there may be present many of the symptoms of passive renal congestion, but there will also be a history of violent pains in the region of the kidneys, of nausea and vomiting, and probably of chills and fever.

Prognosis.—Passive hyperæmia of the kidneys occurring in the course of a cardiac lesion or pulmonary trouble will very probably result seriously. Much depends upon the general condition of the patient, the cause of the congestion, and the accompanying complications. The renal congestion may be greatly relieved, and the patient be comparatively comfortable for a time, but when the congestion is the result of disturbance in the general circulation its presence indicates that the condition of the patient is becoming more serious, and that there is a

probability of the case soon terminating fatally. When the congestion is due to a disturbance in the local circulation, it is possible for the disturbance to be unilateral, and the healthy kidney may perform its function for a considerable time with but little inconvenience to the patient. When the congestion is caused by the pressure of the gravid uterus, it is at once relieved by the removal of the cause, and this should not be delayed if the symptoms become urgent.

TREATMENT.—The treatment of passive congestion of the kidneys depends upon the cause of the congestion. When the disturbance is due to a local cause the treatment is first directed toward removing the cause, and in case that is not possible, means must be used to relieve the symptoms and to build up the general condition of the patient. When the congestion is due to the pressure of the pregnant uterus, it may become necessary to induce labor to relieve the dangerous condition. When the presence of neoplasms in adjacent organs is the cause of the congestion, it becomes the province of the surgeon to determine whether it is possible to secure relief by means of operation.

Renal congestion is usually due to an irreparable malady, and we therefore address ourselves to relieving the urgent symptoms and improving the general condition of the patient in such a manner as to give the most lasting results. As the congestion is most frequently due to some disturbance in the general circulation, the treatment must be directed mainly to the restoration of the disturbed circulation to its normal condition. The means for relieving the congestion are then directed to increasing the cardiac power, to dilating the bloodvessels, or to removing a portion of the blood. Bleeding is not now generally employed for the relief of renal congestion, but the congestion in pregnancy is a very important exception, and bleeding may be resorted in these cases with good results. Cups applied over the region of the kidneys aid in relieving the congestion. When the congestion is due to cardiac failure, the first object should be to restore the heart action, and by increasing its power secure an approximal normal circulation. Rest in bed is of the greatest service.

It is not necessary at this time to discuss in detail the therapeutics of cardiac lesions, but there are a few drugs which act admirably for restoring the heart action and relieving congestion. The sovereign among these remedies is digitalis. It slows and steadies the heart, and under its influence the cardiac muscles seem to regain tone and strength, and the renal circulation becomes re-established. Often an apparently hopeless case, where the heart is failing, rapid, and weak, and the patient is becoming waterlogged and apparently beyond the reach of help, under the influence of digitalis is relieved of the distressing symptoms: the urine becomes more abundant, the albumin disappears, and the patient reaches a comparatively comfortable state. A combination of powdered digitalis and squills, a grain each, and calomel, one grain, given in pill form, one every four hours, will often quickly relieve an intense renal congestion, especially when it is due to cardiac insufficiency. To relieve the arterial tension and to dilate the capillaries nitro-glycerin may be used with advantage combined with the digitalis. A combination of the tinctures of digitalis, strophanthus, and nux vomica often produces a remarkable and excellent result in those cases where the heart is not

able to accomplish its work. Chloral hydrate is often of benefit. In extreme cases sparteine or convallaria sometimes gives good results. The potassium salts may prove valuable with vegetable acids or may be given with the digitalis.

In addition to the use of cardiac stimulants it is important to attend to the general condition of the patient. The regulation of the diet is of the greatest importance. The use of tonics may prove of benefit, and iron helps to overcome the anæmia so common in these cases. When the congestion cannot be relieved by cardiac stimulants, diuretics or hydragogue cathartics should be employed. Vapor baths or the warm bath may be used to great advantage when there is ascites or a tendency to anasarca.

PYELITIS.

By I. N. DANFORTH, M. D.

Catarrhal Pyelitis.

Definition.—A catarrhal inflammation of the pelvis of the kidney—more frequently the right kidney—attended by exuviation of the pelvic epithelium and the presence of leucocytes and papillary casts.

A distinct form of catarrhal pyelitis is not generally recognized in our textbooks, but I long ago satisfied myself, both by clinical and post-mortem observation, that it frequently occurs, although perhaps in the majority of cases it is not diagnosticated until it has degenerated into a suppurative pyelitis.

Etiology.—Exposure to cold and wet; over-exertion, especially lifting and straining; slight injuries, such as kicks or blows over the renal area; lithuria, oxaluria, or the presence of crystals or minute calculi; acute infectious diseases, as pneumonia, typhoid fever, and small-pox; irritations transmitted from the bladder from the presence of calculi or the use of bougies; and especially overdosing with copaiba, sandal oil, and allied remedies,—are the most common causes of catarrhal pyelitis. Many cases occur for which no cause can be assigned, yet a careful inquiry will elicit a well defined cause for the majority.

Pathological Anatomy.—As catarrhal pyelitis is never fatal, very few opportunities for its post-mortem study occur, but I occasionally encountered cases in the post-mortem room when pathologist to St. Luke's Hospital.

When the pelvis is laid open the lining is found to be covered with mucus or muco-pus; when this is washed away the mucous surface appears reddened and injected; the vessels are plainly visible, and many of them tortuous; sometimes ecchymotic patches are seen, and not infrequently irregular areas of ulceration. Sometimes a considerable quantity of gritty or mortar-like material, consisting of mucus or muco-pus mixed with urinary salts, will be found, although such deposits are more common in suppurative pyelitis. The renal structures are usually somewhat hyperæmic, but not markedly so. From this description it will be observed that the lesions are those which are recognized as peculiar to catarrhal inflammations elsewhere.

Symptoms.—The symptoms will depend somewhat upon the cause. In cases produced by cold and wet there will be slight febrile movement, with a temperature of perhaps 100° F.; dry, coated tongue, impaired digestion, perhaps some headache, generally constipation, and dull but not very pronounced or severe pain in the lumbar region. There will be frequent calls to urinate, some cystic irritation, and the urine will be

pale, cloudy with mucus, and will rapidly decompose and become offensive. Cases dependent upon the irritative effects of urinary salts will be attended with much irritation of the bladder, with a scalding sensation along the urethra during urination, and the patient will be annoyed by frequent and imperative calls to urinate. The urine is usually strongly acid, of high specific gravity, and upon standing throws down a copious grayish precipitate composed of mucus, lithic acid, or calcic oxalate, and certain morphological elements to which I shall allude presently. The patient will complain of pain in "the small of the back," and frequently the erector spinæ will show slight tenderness. Digestion will generally be poor, the appetite whimsical, the tongue coated, the breath foul, the bowels torpid, and the digestion, with other concurrent symptoms, will indicate an inactive liver. In cases which occur during the course of acute infectious diseases the symptoms are usually masked by the more urgent and outspoken symptoms of the acute illness, but a study of the urine will reveal the characteristic crystalline and cell deposit. Severe attacks of catarrhal pyelitis are sometimes the consequences of over-dosing with balsam, etc. in the treatment of urethritis. In such cases the backache will be pronounced, the urine will become ropy with mucus, perhaps slightly tinged with blood, and a heavy deposit of thick, ropy mucus, mixed with crystalline particles, will fall. The pyrexia and gastro-enteric symptoms will be more pronounced than in milder cases. Prolonged disease of the bladder, from whatever cause, is likely to induce pyelitis, but the bladder symptoms will quite overshadow those of pyelitis. A study of the urine will invariably reveal the facts.

The urine in catarrhal pyelitis is generally rather abundant (four pints or more in twenty-four hours)—is somewhat pale and cloudy with mucus, which can be seen floating in shreds or patches. The reaction is usually acid, the specific gravity rather low (1010 to 1015), and albumin, in quantities varying from a barely appreciable trace to a tenth of a gramme per litre, may be found, although it may be entirely and permanently absent. The microscope shows patches of "transitional" epithelium in various stages of cloudy swelling, numerous leucocytes or cells resembling them, and usually some characteristic bodies which I have denominated "papillary casts," because I believe them to be the casts derived from the papillary portion of the straight tubes. These casts are at least three times as large as the largest tube cast of Bright's disease, and are composed of a group of columnar epithelial cells arranged in a linear or cylindrical form, being held together by intervening mucus. As I have frequently dislodged similar groups of cells from the papillary portions of the straight tubes by squeezing the papillæ, I am convinced that the casts above described are derived from the same source, and that they go far toward establishing a diagnosis of catarrhal pyelitis.

Complications and Sequelæ.—Catarrhal pyelitis is not especially liable to concurrent complications, with the exception of cystitis. Lithuria or oxaluria, which so frequently precedes pyelitis, cannot be regarded as a complication, but stands in the relation of a cause.

As logical sequelæ of catarrhal pyelitis I may enumerate suppurative pyelitis, renal calculus, chronic tubal or interstitial nephritis, and

it is not unlikely that tuberculosis renalis may be induced or invited by the presence of catarrh of the pelvis.

The disease in question may without doubt prove the entering wedge which leads to the development of pyonephrosis, as I shall point out when treating the latter. Indeed, many a kidney may date its ruin from the advent of catarrh of the pelvis.

DIAGNOSIS.—The differential diagnosis of catarrhal pyelitis presents no difficulty if the practitioner is alive to the fact that it is likely to be present. Pyelitis may be masked or stifled by the more loudly complaining bladder, for cystitis is almost sure to accompany pyelitis, and the symptoms of cystitis are always pronounced, while the symptoms of catarrhal pyelitis are generally feebly marked. But the characteristic backache, frequent urination, acid urine, abundant mucus, lithic acid deposits, and the presence of the above mentioned "papillary casts" will establish the diagnosis.

PROGNOSIS.—Many cases develop, run their course, and recover spontaneously unrecognized, and in my judgment this class includes the greatest number.

Many other cases are recognized, are properly and successfully treated, and recover in a few weeks or months. But quite too many go unrecognized and untreated, and they result in suppurative pyelitis, renal calculus, pyonephrosis, or some other lesion equally grave. If every case of catarrhal pyelitis could be promptly recognized and properly treated, comparatively few cases of suppurative or calculous disease of the kidney would occur.

TREATMENT.—A plain and simple diet, consisting largely of milk; the plentiful use of pure water; abstinence from alcoholic stimulants and the avoidance of exposure to cold and damp,—these constitute the chief factors in the successful treatment of catarrhal pyelitis. Constipation should be remedied by the use of salines, and digestion aided if necessary—as it generally will be—by pepsin or some equivalent. If the urine is highly acid and loaded with lithic acid or its compounds, citrate of potash or of lithium may be given, the granular effervescing form being preferable. When the urine is loaded with adhesive ropy mucus, I have found copaiba, Canada balsam, and oil of sandalwood very efficient. Either one may be given in capsules containing not more than five minims each, and I usually direct one capsule to be taken every three hours and as far from meal-times as possible. The remedy selected should be continued for several weeks without change, unless some special reason arises for discontinuing, the object being to get its alterative effect.

Although the terebinthinate remedies in over-doses, as they are frequently given, produce nephritis or pyelitis, it is nevertheless undeniably true that in small doses they are of great value in the cure of the same diseases. But continuity of treatment and small doses are only the conditions upon which success can be expected.

Triticum repens, stigmata maidis, and the saw palmetto are each very useful, and may be given in infusion or fluid extract. If the infusion be given, one ounce of the drug to the pint of water should be employed, and the patient may take one pint of the infusion of either per diem, in divided doses. If the fluid extract be selected, the dose

will be one drachm four times a day, in water. If the patient complains of pain or of much cystic irritation, it is well to give an occasional dose of codeine alone or combined with other remedies. Another remedy of much value is benzoic acid, in doses of ten grains four times a day. Codeine may be combined with the benzoic acid if it is indicated. A formula which I have employed long and successfully in the treatment of catarrhal pyelitis and the almost inevitably consequent cystitis is the following:

℞. Tincturæ opii camphoratæ,
Extracti tritici fl.,
Extracti stigmatæ fl.,
Syrupi althææ, āā. ℥ij.—M.

Sig. A dessertspoonful four times a day in half a glass of water.

This combination is especially valuable in the early stage of pyelitis when the lumbar pain and cystic irritation are urgent.

Suppurative Pyelitis (or Pyonephrosis) and its Sequelæ.

Under the above title I propose to consider several lesions of the kidney which are attended by suppuration, which have essentially the same origin, and ultimately reach the same end. These are (1) Suppurative pyelitis; (2) Pyelo-nephritis; (3) Suppurative nephritis. As these are practically varieties of a single morbid process, they may be defined as a suppurative process, generally secondary to some disease of the pelvic organs which obstructs the exit of urine, thus producing distention of the ureters and pelvis renalis; they may be produced by secondary infection from a diseased urethra, prostate gland, or bladder, or the infection may occur during the progress of an acute infectious disease, or they may follow simple catarrhal pyelitis.

As its name implies, this is a suppurative inflammation involving the pelvis of the kidney, including the calyces and generally the papillary extremities of the straight tubes. As regards gravity, causation, and results, it must be sharply distinguished from catarrhal pyelitis, although the latter may, and not infrequently does, lead to the development of the former.

Etiology.—In nearly all cases of suppurative pyelitis two etiologic factors are present—namely, obstruction and infection. Obstruction may arise from enlargement of the prostate, stricture of the urethra, phimosis, paralysis of the bladder, calculi, blood clots, or other obstructive agents in the ureter or the pressure of morbid growths upon the ureter, and in the female tumors of the ovaries or uterus or inflammatory exudates encroaching upon and compressing the ureter. In short, anything which hinders the outflow of urine, thereby causing retention and gradual dilatation of the renal pelvis, acts as a predisposing cause of pyelitis. Infection may arise from gonorrhœal urethritis or cystitis, suppuration of the prostate gland, the use of unclean catheters or bougies, suppurative cystitis consequent upon stone in the bladder, or a dilated or irritated pelvis may become infected through the agency of the acute exanthemata, carbuncle, pyæmia, diphtheria, erysipelas, or scurvy. But now and then a case occurs in which neither

infection nor obstruction can be detected, although one or both may exist in so slight a degree as to escape detection.

Pathological Anatomy.—The renal pelvis is dilated, sometimes very greatly, sometimes but slightly; its walls are thickened and indurated; the ureter is generally trumpet-shaped, and also thickened and elongated; upon opening the pelvis its surface is seen to be rough, sometimes ulcerated, its vessels are large and tortuous, and it is frequently occupied by a thick mortar-like deposit made up of decomposed urine and pus and urinary "gravel;" small calculi are sometimes ensconced in the calyces; the papillæ are flattened and pushed back by the continuous pressure of the accumulating pus and urine. The renal cortex and medulla are not markedly changed, beyond being hyperæmic or perhaps slightly indurated.

Symptoms.—A pronounced case of suppurative pyelitis is usually ushered in by a sharp rigor, followed by rapidly rising temperature and perhaps free diaphoresis, and these events sometimes become so periodic as to suggest malarial fever. In a recent case involving the right pelvis, which occurred seven weeks after left nephro-lithotomy, the pyrexia assumed a distinctly quotidian type, the morning temperature being normal, the afternoon temperature being 103° F. Along with or immediately following the pyrexia there will be a dull "aching" pain on the affected side, accompanied by marked tenderness upon bimanual pressure. The area of renal dulness will be increased. The urine will become turbid or perhaps deeply tinged with blood; pus and albumin will appear from six to twelve hours after the initial rigor; and partial suppression may occur. The microscope will show pus corpuscles, blood globules, swollen pelvic epithelium, and probably linear groups of cells from the papillary tubes. In some cases minute blood clots will be found or perhaps elongated cylinders of clotted blood, these cylinders having lingered long in the ureter. If the hemorrhage be only slight, the blood is apt to be dark and smoky when passed; if in large amounts, it will be fresh and bright.

Pyelitis may be complicated or followed by pyelo-nephritis or suppurative nephritis (multiple abscess of the kidney). It will also be quickly followed by cystitis, due undoubtedly to the irritating character of the urine escaping from the faulty kidney. Sometimes the symptoms of cystitis are so violent and aggressive that they obscure the symptoms of pyelitis and lead the practitioner astray.

Diagnosis.—Pyelitis in the early stage requires to be differentiated from renal calculus, perinephritis, pyelo-nephritis, and suppurative nephritis; and it is always wise to seek for symptoms of pyelitis in cases of cystitis of doubtful origin, since the latter is so often a consequence and sequel of the former.

The symptoms of renal calculus are quite different from those of pyelitis. The sharp, acute pain coming on in paroxysms, especially after exercise; the distinctly localized, or, as I am accustomed to designate it, "focalized," character of the pain, so that the patient can point to its exact position; the slight amount of blood in the urine, and its certainty to follow each severe paroxysm of pain; the small amount of pus or even its entire absence; and the long duration of these paroxysmal attacks without serious inroad upon the health, together with the marked

absence of the distinctive symptoms of pyelitis already described,—will be sufficient to eliminate renal calculus from a case of idiopathic pyelitis. It must be added, however, that a calculus when it falls into the pelvis and remains loose therein is very likely to induce pyelitis, when the symptoms thereof will be added to the pre-existing symptoms of calculus.

Perinephritis may be differentiated by the larger area of pain and its refusal to submit to definite metes and bounds, by the more pronounced and more superficial tenderness on pressure, and by its predilection for the lumbar region; by the inability of the patient to stoop, sit down, or get up without great pain, and by his disposition to "lean" so as to favor the affected side, as in psoas abscess; and by the absence of pus, blood, broken-down tissue, and albumin in the urine. Of course pyelitis and perinephritis may occur together or one may follow the other, in which case the symptoms of both will be present and will be recognized by a careful diagnostician.

Pyelitis is so frequently followed by or culminates in pyelo-nephritis that a refined differential diagnosis is impossible, and never essential from a clinical point of view; but the latter is distinguished by a greater degree of tenderness over the kidney, a greater quantity of pus, more albumin, the presence of much degenerated renal epithelium, and usually large granular tube casts in the urine—a more persistent pyrexia with a decided tendency toward "hectic." The same observations apply to the condition known as suppurative nephritis or multiple abscess of the kidney, except that the symptoms of the latter are rather more acute and aggressive, and septicæmia is apt to appear more promptly and throw the patient into a position of gravity and danger.

Prognosis.—An uncomplicated case of suppurative pyelitis is not a disease of such gravity as to call for a grave prognosis. If the diagnosis is established early and an appropriate plan of treatment is adopted, recovery generally follows, although a rapid recovery is impossible. Several weeks, and probably several months, of continuous treatment will be required before the case can be dismissed. When the renal structure is involved and the case becomes one of "pyelo-nephritis," the chances of recovery are greatly lessened; in fact, surgical aid will almost invariably be required before any radical results are reached. Many cases recover, however, after lumbar incision, irrigation, and drainage.

Suppurative nephritis or multiple abscess of the kidney always brings danger in its train, many cases being quite beyond the reach of physician or surgeon. It is happily a rather rare complication of pyelitis, and if the latter is promptly recognized and efficiently treated can generally be prevented. Cases which commence as primary infective suppurative nephritis are always very dangerous, and generally fatal from acute and aggressive septicæmia.

Treatment.—The first requisite is rest—in the acute form absolute rest in bed; in subacute and chronic suppurative pyelitis confinement to bed is not necessary, but the patient should lead a quiet, secluded life, much of the time in the recumbent position. Gentle exercise to a limited extent may be permitted in favorable weather, but the tendency to

overdo must be restrained. An appropriate diet is very important; meat should be restricted to a small amount once a day, fish or fowl being given the preference over beef and mutton. Milk is the ideal food, and with some display of culinary resource it can be prepared in a variety of palatable forms. Fruit and vegetables may be allowed in reasonable quantities. The patient should drink freely of pure water, but he should abstain almost wholly from alcoholic beverages. He should also dress warmly, carefully anticipating, so far as possible, changes in the weather, so as to avoid chills and storms.

Two indications present themselves for therapeutic solution: first, to render the urine plentiful and bland, and secondly, to so medicate the urine with alterative remedies that the inflamed pelvis may be gradually won back to a state of health.

The first indication is easily met. Copious draughts of barley, rice, or toast water, holding in solution a drachm of potassic citrate to the pint, should be given; this will increase the volume of urine, render it bland and unirritating, while the alkaline salt will prevent the precipitation of the uric acid and urates in the diseased renal pelvis. This treatment ought to be continued without interruption until the urine becomes copious and the muco-pus has wellnigh disappeared. Gum water (acaciæ ℥j, aquæ Oj) or any other bland diluent drink may be used as a vehicle for the potassic salt in place of those already mentioned. Potassic carbonate is sometimes preferable, especially if the patient be rheumatic and the lithic acid tendency be unusually pronounced. In cases where the lithic acid deposit, pure and simple, seems to be the pathological pivot the salts of lithium (lithic citrate or lithic carbonate) are very useful, although in my experience the potassic citrate is quite as efficient. Whatever saline is given, two practical points should not be overlooked—namely, first, the medicine should be freely diluted in some bland infusion, like those already mentioned, and secondly, it should always be given when the stomach is empty, so that it may be rapidly absorbed without essential change.

The second indication, that which calls for the administration of "alterative" remedies, is met by the use of such agents as triticum repens, stigmata maidis, saw palmetto, and the purely balsamic remedies. They must be given precisely as I have indicated under the head of Catarrhal Pyelitis—namely, in small doses and continuously. They must be given purely as "alteratives." An alterative is a remedy which acts slowly, continuously, and persuasively; it gently wins a diseased organ back to a state of health or an approximation thereto by an influence which is not perceptible from day to day, but can be appreciated only after the lapse of weeks or even months. Hence alterative remedies must be used for long intervals, and they should almost invariably be administered when the stomach is not engaged in the process of digestion.

Pyelo-nephritis.

When suppurative nephritis does not result in recovery, it is almost certain to proceed a step farther and involve the substance of the kidney. It is then known as "pyelo-nephritis," or suppuration of both

pelvis and parenchyma. It depends upon the persistence or increase of the same causes which produced the initial pyelitis. The pelvis is gradually dilated, the papillæ pushed back, the calyces are partially obliterated, the interpyramidal connective tissue is gradually thickened, the capsule develops into a thickened, brawny, indurated sac, and the kidney is at last converted into a series of wedge-shaped abscesses, each abscess representing a former pyramid or lobule. This process of progressive destruction is sometimes so complete that only a very thin layer of cortical substance remains, and I have seen one case in which not a vestige of renal structure could be detected, the kidney having been actually replaced by symmetrical wedge-shaped cavities filled with pus. The kidney is generally considerably enlarged, although the process of destruction may proceed from beginning to end without enlargement; in fact, if the pus finds exit through the ureter to any considerable extent, the kidney is likely to diminish rather than increase in size.

Symptoms.—The symptoms of pyelo-nephritis are those of pyelitis emphasized and intensified. Digestion will be much disturbed; the tongue will be furred; the patient will complain of a sense of weariness and lassitude; the morning temperature will rarely be less than 100°, while the evening temperature will be likely to reach 101° or 102° F. Slight rigors are quite common, and occasionally a pronounced chill will be experienced. A constant, dull, and sometimes throbbing pain will be felt in the affected kidney; a well defined area of renal dulness can generally be detected on percussion, and the affected organ is always tender—sometimes acutely so—to the touch. The urine will be loaded with pus, generally more or less offensive, and shreds of broken-down tissue and degenerated epithelia will be present. Crystals of phosphates, urates, or oxalates in groups or masses will sometimes be found, but they are not constant.

The diagnosis presents no difficulties. The general malaise, the persistent temperature, the constant pyuria, the localized pain, the renal enlargement, tenderness, and probable fluctuation, together with the previous history of the case, cannot fail to conduct the practitioner to a correct diagnosis.

In former years the prognosis of pyelo-nephritis was always grave, as no methods of internal medication promised any relief; but since the development of renal surgery with its brilliant results all this is changed. The prompt performance of lumbar nephrotomy, with free irrigation and drainage, will result in radical cure in the great majority of cases. No unnecessary delay should be allowed after the diagnosis has been established. The operation should be done at once, the kidney freely incised along its convex border; every pus cavity should be sought out and thoroughly evacuated, and subsequent irrigation and drainage should be thorough and painstaking.

Treatment.—I have had most satisfactory results in my operations for the cure of pyelo-nephritis—results which I attribute to promptitude in operating and thoroughness in after treatment.

During convalescence the patient will need substantial nourishment and active tonic treatment. For the first few days following the operation it is well to give ten grains of quinine a day—preferably in two

forenoon doses—as there is likely to be a lingering septic fever which the quinine will counteract. A little later quinine and iron may be combined (℞. Quininæ sulphatis, gr. ij ; Ferri citratis, gr. iij), and given three times a day. Now and then a case occurs in which the kidney is so thoroughly destroyed and broken down that nephrectomy is the only resource. While this adds materially to the danger, it does not by any means render the case hopeless. I have had several successful cases of nephrectomy for suppurating kidney, and regard the operation as a comparatively safe and easy one. David Newman gives a list of 44 lumbar nephrectomies for suppurating kidney, with 32 recoveries and 12 deaths. This is a very encouraging result, and fully warrants the performance of the operation in cases where no other resource remains, although the milder operation of nephrotomy should be selected unless nephrectomy is imperatively demanded.

Suppurative Nephritis.

Definition.—Beck's definition of suppurative nephritis—"acute interstitial nephritis with scattered points of suppuration"—scarcely goes far enough. It seems rather to be a general acute suppurative process, involving all the tissues of the kidney, so that it is not only "interstitial," but it invades the tubules, the capsule of Bowman, and even the bloodvessels. Very rarely it occurs without antecedent pyelitis, but the great majority of cases are secondary to pyelitis and are due to direct infection derived therefrom. But, whether associated with pyelitis or not, it is always the result of acute and virulent sepsis, which spreads desolation in the affected organ. The kidney is rapidly converted into a congeries of minute abscesses which vary in size from a pin's head to a marble. So rapid is the process that in forty-eight hours an apparently healthy kidney may be honeycombed by hundreds of suppurating foci. These foci are apt to enlarge, coalesce, and finally burst into the renal pelvis or through the capsule into the perirenal tissues. In either case the kidney is rapidly but effectually ruined.

Cases occurring without antecedent pyelitis (which stands in the relation of cause), but which are induced by sepsis derived from some acute germ disease—as, for example, smallpox—will generally present bilateral suppurative nephritis, since the efficient cause will affect both kidneys alike.

Symptoms.—The symptoms of acute suppurative nephritis at once indicate its gravity. A severe rigor followed by high temperature (103° to 105° F.), headache, slight but increasing hebetude or a mild inoffensive delirium, nausea and vomiting, dry brown tongue, and a strained and anxious countenance, at once proclaim the gravity of the situation. The pain located in the kidney, which is generally a throbbing, pulsating pain, the tenderness on pressure, the increased and rapidly increasing area of dulness, the thick, scanty, and perhaps bloody or purulent urine heavily clouded with albumin, together with the prior history of pyelitis, form a picture sufficiently vivid to guide the practitioner to a correct diagnosis. In a case involving both kidneys, complicated by the existence of some acute infective disease and *not* preceded by pyelitis, a little confusion might arise. But in such a case

there would be almost total suppression of urine and the pain and tenderness would extend to both kidneys. And the very fact that renal pain and tenderness, hæmaturia, pyuria, and albuminuria occur during the progress of an acute infectious disease should at once direct the attention of the attending physician to the kidneys. In a case of acute suppurative nephritis, where the pus breaks through the capsule and infiltrates the perirenal tissues before the physician is called, it might at first glance be mistaken for primary perirenal abscess; but the very gravity of the symptoms, the evident extreme illness of the patient, together with the characteristic appearance of the urine already described, ought to awaken the physician to an appreciation of the real facts of the case.

In a true case of infective nephritis with suppurating foci the prognosis is very grave. The great majority of cases prove fatal. Now and then one survives, and after a long and tedious convalescence recovers fully—not as the result of treatment, but by means of an inherent vitality and unusual recuperative power.

Treatment.—The treatment of suppurative nephritis must depend much upon the stage at which it is first seen. If a case is seen early and the diagnosis can be satisfactorily established, it will be well, after sending the patient to bed, to apply three or four leeches over the affected kidney, or over both kidneys if necessary, to be followed by hot fomentations. If, however, the symptoms are not very pronounced, but suppurative nephritis seems probable, cold applications may be preferable. Quinine should be given in full doses, not less than five grains every four hours, and, if symptoms of collapse threaten, camphor, ammonia, strychnine, and perhaps alcoholic stimulants. Some benefit may be derived from antiseptics administered internally, the most efficient being guaiacol carbonate in doses of five grains every four hours, or creasote carbonate in similar doses. The main point is to select an antiseptic of undoubted potency, and administer it up to the limit of toleration. I am of the opinion that quinine and guaiacol are "synergists" in regards to each other, and the same is true with respect to quinine and creasote; hence in a grave case of septicæmia I advise alternating the antiseptic selected with full doses of quinine.

If fluctuation can be detected, even though it seem deep and obscure, the aspirating needle should be introduced, and if pus be found the attempt should be made to reach it by incision. If free drainage and antiseptic irrigation can be established, it will certainly improve the patient's chances. If superficial fluctuation and tumefaction appear in the lumbar region, indicating the perforation of the renal capsule and the escape of pus into the perirenal tissues, a free incision should at once be made so as to ensure thorough irrigation and perfect drainage. Symptomatic indications, such as weak heart, constipation, nervous exhaustion, etc., must be met and combated as they arise.

RENAL CALCULUS; HYDRO-NEPHROSIS; RENAL TUMORS; RENAL ABSCESS; PERI-RENAL ABSCESS; RENAL PARASITES; NEUROSES OF THE KIDNEY.

By I. N. DANFORTH, M. D.

RENAL CALCULUS.

Renal calculi are by no means uncommon. It is wellnigh certain that minute concretions are constantly forming in the kidneys of very many people, and that they are passed unconsciously or with a slight and momentary sense of urethral discomfort or irritation. Many patients learn to recognize the early symptoms of "a fit of the gravel," and they understand that these symptoms are pretty certain to be followed by the passage of a shower of coarse sand or by a few minute concretions, and that the passage of either is followed by immediate and perfect relief. I have many specimens of these little calculi, from the size of a pin's head to that of a pea, which have been brought to me by patients. The very minute calculi are passed with little trouble or distress, often, as I have already said, unconsciously. When they are too large to pass readily through the ureter, they are liable to cause acute, sometimes agonizing, pain during their journey to the bladder. They are often detained in the kidney until they become so large that their transit through the ureter is impossible; then there remains but one resource—namely, removal by "nephro-lithotomy," to which the patient generally submits after years of weary suffering.

Etiology.—It is impossible in this place to discuss at length and in all its bearings the pathology or essential causes of renal calculi. Moreover, it must be admitted that some important questions closely related to the evolution of renal concretions are not yet settled, and will not be until physiologists have dipped deeper into the mysteries of assimilation and disassimilation.

But in any case of "gravel," as it is called when the crystalline deposit appears as loose sand, or of renal calculus, as it is called when the "gravel" is united into masses from the size of a pin's head upward, two etiological elements must concur—namely, a state of the system which we somewhat blindly call a "diathesis," in consequence of which certain urinary factors appear in excess of their normal amount, and a state of the secreting structure of the kidney which invites a deposit or crystallization of these over-abundant factors in the renal tubules. As an example of the state of system alluded to I may mention the condition known as "lithæmia," in which lithic acid ap-

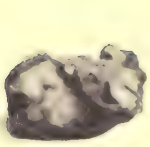
pears in such excess that the water of the urine is unable to hold it in solution, and it is precipitated in the renal tubules. But there is also a concurrent disturbance of the renal structure which favors the precipitation of the urinary crystals, and this is the first step in the formation of calculus. This disturbance is usually a slight catarrhal inflammation of the renal tubules and their epithelia, resulting in cloudy swelling of the latter, the formation of tenacious mucus, and narrowing of the lumen of the tubules, thus retarding the flow of urine and inviting precipitation of the urinary solids. As a further step there is formed in the kidney, according to Scharing and Ord, a "colloidal substance," the "intervention of which is necessary to the formation of calculi," and Scharing believes that "the nuclei of calculi are bound together or enveloped by mucus, albumin, or other organic matter, which entangles the chemical constituents and determines their form of crystallization" (Newman). This "colloidal substance" and the "mucus, albumin, or other organic matter" are probably one and the same thing. For years past I have observed as a clinical fact, and have taught in my lectures, that gravel and renal calculi are invariably accompanied and probably preceded by renal catarrh, as evidenced by cloudy urine, the presence of leucocytes, swollen epithelial cells, cylindroids or mucin casts, and frequently a trace of albumin. Moreover, the cylindroids often contain crystals of lithic acid or calcic oxalate in their midst, which shows that the crystals were formed in the renal tubules, and that they were entangled in the cylindroid during its formation. I am therefore persuaded that the catarrhal kidney is an almost necessary precedent to a renal calculus, and this is especially true of lithic acid and oxalate of ammonia gravel and calculi. According to Mr. Henry Morris, the "most common form of renal calculus is the uric acid; the next most common form is the oxalate of lime." These are the forms of "primary calculus." Stratified calculi are frequently found composed of alternate layers of lithic acid, calcic oxalate, and mixed phosphates. Occasionally a little mass of dried blood forms the nucleus of a renal concretion. Cystine calculi are very rare. I have but two specimens in my own collection, and both of them were passed by the same patient at an interval of about one year.

Renal calculi are found at all periods, from intra-uterine life up to old age, but they are most common in the children of the poor and in the well-to-do beyond fifty years of age.

According to Mr. Thomas Taylor, the nucleus of infantile calculi is urate of ammonium; the nucleus of calculi in the kidneys of young adults is lithic acid; the nucleus of calculi found past middle life is calcic oxalate (Morris). Nevertheless, I have removed a calculus of nearly pure calcic oxalate from the kidney of a young woman of eighteen, and the history pointed clearly to its having existed not less than four years.

Calculi may exist in one or both kidneys; one or several calculi may form in the same kidney; they vary in size from a pin's head to masses which completely fill the pelvis and calyces; they vary greatly in form, some being smooth and rounded, others rough and branched or pronged in the most fantastic manner. But this curious antler-like branching is much more easy to understand since Mr. Jordan Lloyd has demonstrated the true architecture of the renal pelvis and calyces by means of paraf-

PLATE XV.



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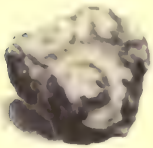

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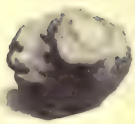

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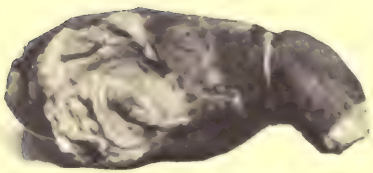

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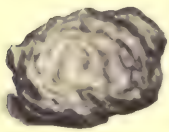

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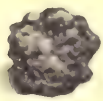

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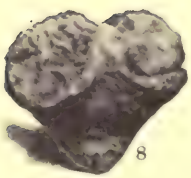

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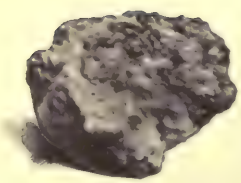


9

Renal Calculi Removed by Lumbar Nephro-Lithotomy.

1, 2, 3, **Phosphatic Calculi from Kidney, greatly distended by Pus.**

4, 5, **Stratified Calculi removed from Right Kidney of woman aged 30; the Kidney contained 16 ounces of Pus; the long horn-like process on No. *4* reached down into the ureter, almost closing it.**

6, **Small Oxalate of Lime Calculus from Kidney of woman aged 23.**

7, **Lithic Acid Calculus from Kidney of woman aged 24; this Calculus produced intolerable pain and considerable hemorrhage.**

8, 9, **Calculi Composed of Mixed Urates from Kidney of a man aged 65; the Kidney was a mere pus cavity, and the patient was much broken down by pain and sepsis.**

fin casts. The accompanying Plate XV. represents very accurately a variety of renal calculi.

Symptoms.—The symptoms of renal calculus depend upon the size, variety, locality, and mobility of the concretion; consequently, the symptoms will vary greatly in different cases, and the diagnosis of a calculus will always be to a considerable extent a matter of conjecture. Minute calculi are frequently passed through the entire urinary tract with so little distress as scarcely to attract the patient's attention. There may be slight intermitting attacks of pain as the stone traverses the ureter and some irritation of the urethra as it passes through that canal, but the symptoms are not sufficiently pronounced to require medical aid, and so the patient passes a calculus without knowing it.

When the calculus attains a little larger size—say, as large as a bean and not unlike it in form—its passage from the kidney to the bladder is attended by very pronounced symptoms. First, the patient will complain of brief yet quite sharp attacks of pain, which he will locate quite accurately in the region of the kidney, most frequently the right kidney. The pain generally comes on suddenly and unexpectedly, usually after some active exercise, as lifting, jumping, or running. The patient generally will remember, however, that for some months previously he has experienced now and then sudden sharp "pricking" pains, brief in duration and not of great severity, but always located in the same place. Some patients will at this stage have slight hæmaturia, but it is the exception rather than the rule. These symptoms are produced by small calculi of the lithic acid or calcic oxalate variety which have found their way into the renal pelvis and are lying loose therein. Sooner or later these little calculi enter the funnel-shaped opening of the ureter and commence their journey to the bladder. Then begins a second series of symptoms which are so pronounced that their import can scarcely be misunderstood. After a sudden strain or jump or fall, or perhaps without any such antecedent, the patient experiences a sharp, decisive cutting pain, which obliges him to seek his bed and frequently causes him to cry out with agony. He locates the pain very accurately over the renal pelvis. He bends his body forward and flexes his limbs, so as to relax the abdominal muscles as much as possible. He writhes and twists uneasily, calls testily and impatiently for relief, and gives every evidence of suffering the most acute torture. I have seen strong and resolute men cry out with agony and amid sobs and tears beg piteously for help. Meantime, there are likely to be nausea and vomiting, a coated tongue, anorexia, moderate pyrexia (101° to 102°F.), and considerable reflex nervous disturbance, as evinced by rapid, irregular breathing, quick, excited pulse, occasional transitory rigors, and sometimes an hysterical loss of mental balance. The terrible pain will present two characteristics: first, it will be paroxysmal, the paroxysms lasting from a few minutes to several hours; and secondly, it will change its location from time to time, passing in an oblique direction from the renal pelvis toward the bladder. It will be observed also that the rapidity of the migration will be pretty accurately determined by the frequency of the paroxysms of pain, the paroxysm being caused mainly by the spasmodic contraction of the ureter in its efforts to propel the calculus toward the bladder. There will usually be frequent and urgent calls to urinate, with consid-

erable cystic tenesmus, but the quantity of urine voided will be small, and it will frequently be colored with blood or it may contain minute blood clots or long cord-like clots which have formed in the ureter, the hemorrhage being the result of laceration of the ureter by the rough surface of the calculus. There will also be paroxysms of acute burning or stinging pain in the glans penis, as well as pain in and retraction of the testicle of the affected side. The urine contains a small amount of albumin, many leucocytes, red blood globules, and sometimes patches of epithelial cells. It is also usually cloudy with mucus, and during the passage of a stone it contains a large quantity of thick, sticky mucus more or less stained with blood. Pains are frequently felt in the leg of the affected side, and Dickinson mentions cases where a burning pain was felt on the sole of the foot. After a period of time lasting from a few hours to three or four days the symptoms suddenly cease, and the patient experiences a sense of most welcome and grateful relief and comfort. This is due to the fact that the stone has completed its journey through the ureter and has entered the bladder. In most cases the sufferer can determine by his own sensations when the moment of relief comes. The sudden and total cessation of pain, the general relaxation of the overstrained tissues, and the confident feeling of comfort assure him that his sufferings are over, at least for the present, and a few hours later he resumes his usual occupation, perhaps to pass through the same experience again in a few weeks or months.

If the calculus is retained in the kidney, the symptoms will depend upon its location and mobility. If it is encysted in the cortex, and is therefore fixed and immovable, no pronounced symptoms may be manifested; in fact, it may remain harmless and painless, and therefore quite unsuspected, until its possessor dies of some disease wholly unconnected with the kidney, and the presence of the calculus may never be known. Or it may be accidentally discovered if a post-mortem examination is made. I have twice found large encysted calculi post-mortem which had produced no ante-mortem symptoms, and were therefore unsuspected. Generally, however, an encysted calculus will cause some dull, deep, but ill-defined backache, and the patient will have worn plasters or rubbed himself with liniments without suspecting why. But a calculus which is lying loose in the renal pelvis or in a cavity in the cortex makes its presence known by very positive symptoms. A deep, dull, aching, sharply localized pain, succeeded now and then, and especially after exercise, by a sharp, cutting, piercing pain, which extends over a small area or is, as I am accustomed to designate it, "focalized," is the first and most uniform symptom. Hardly any day goes by without more or less pain, but it is not very severe, and does not disable the patient, although it frequently leads him to regulate his movements with thoughtful care, and especially to avoid jumping, running, or any violent exercise. If he indulges in any athletics or lifts, or strains himself in any way, he is almost sure to suffer a sudden seizure of acute intense pain, which covers so small an area that the word "local" is hardly accurate enough, and hence I have designated these peculiar pains as "focalized" pains. There is a merciless sharpness, acuteness, and intensity characterizing the pain produced by renal calculus which is well-nigh pathognomonic; the patient is able to locate it exactly, and he will

frequently assert that the painful spot is not larger than the tip of his finger.

Another important symptom of renal calculus is hæmaturia. The blood is usually rather small in quantity; not constant, but most likely to appear after some quick, sudden jarring motion of the body; it almost always accompanies or follows immediately after the acute pain which I have just described; it is generally fresh and bright, although it may be black and grumous, and frequently minute coagula or long, slender cylinders are discharged. Microscopic examinations of the urinary sediment will often reveal the presence of blood globules where no blood is visible to the naked eye. The urine is constantly turbid with mucus and muco-pus, generally contains a little albumin, and the microscopic examination will show renal epithelia, leucocytes, or pus cells, and sometimes tube casts, showing that some portion of the renal parenchyma is in a state of exudative inflammation. There is generally considerable irritation of the bladder, resulting in frequent micturition, pain, and cystic tenesmus, and the bladder symptoms are sometimes so urgent that they mask the renal symptoms, and direct the physician's attention away from the real point of trouble. If the case is one of long standing, the renal pelvis will be in a state of suppurative inflammation, and, quite as likely as not, suppurating cavities will be found in the parenchyma. Under these conditions symptoms of sepsis, such as rigors and intermitting pyrexia, will be present. The affected kidney may not be enlarged at all, or it may be distended with pus and mortar-like deposit to double its natural size. It may be very greatly damaged by the formation of multiple pus pockets and the consequent indurating inflammation, or the injury may be so very slight as to be scarcely apparent. In 3 of my cases of nephro-lithotomy the kidneys were practically uninjured, and in 1 of them the symptoms could be traced back with perfect clearness for twenty years. In nearly every case the kidney will be tender to pressure, and the tenderness is of a peculiarly pungent type, which causes the patient to shrink with a sudden start when bimanual compression is made.

Diagnosis.—An absolute diagnosis of renal calculus cannot be made; yet a fairly reliable "working" diagnosis is possible in the majority of cases. Excessive lithæmia or oxaluria, commonly known as "gravel," and tuberculosis and sarcoma in their early stages, may so closely resemble renal calculus in their semiology as to perplex the most astute diagnostician. Lithæmia, when persistent and excessive, may cause pain and hæmaturia and all other symptoms of calculus. I have seen several such cases. But the pain is less severe; is not so distinctly paroxysmal; is not so defined and local; is usually accompanied or followed by a lithic acid "shower," after which the symptoms promptly disappear. The blood is small in amount, just enough to plainly color the urine. Thus the symptoms, while resembling those of calculus, are so much milder in type that an accurate conclusion can generally be drawn. At the same time, it must be remembered that attacks of acute lithæmia may occur when a calculus is actually present, so that a "lithic shower" with its characteristic symptoms does not disprove the existence of calculus. In fact, repeated attacks of lithuria might alone arouse suspicion of calculus, and these observations

apply with equal force to oxaluria. If relief follows treatment for lithuria or oxaluria, it will strongly indicate the absence of calculus.

It is always extremely difficult to distinguish the early state of renal tuberculosis from renal calculus. Yet there are certain points of difference which will enable the careful observer to make a reasonably certain diagnosis. Renal tuberculosis is rarely primary; hence a tuberculous kidney is usually preceded by some other tuberculous deposit. The pain of tuberculosis is, when carefully analyzed, unlike that of calculus in that it is more continuous, less paroxysmal, and not "focalized;" moreover, it does not shoot down into the bladder—does not produce frequent micturition or retraction of the testicle. The hæmaturia of the early stage of tuberculosis is generally small in quantity, but persistent rather than paroxysmal, and not infrequently it is absent until the disease is well advanced. In renal tuberculosis the urine contains comparatively little sediment until the disease is well advanced, while renal calculus generally causes a copious sediment, which has already been described. The urinary sediment should always be examined for tubercle bacilli in suspected cases—in fact, in every case of persistent pyuria of renal origin. Of course the presence of the bacillus tuberculosis at once establishes the diagnosis of tuberculosis somewhere in the genito-urinary tract, and with the symptoms above detailed almost certainly in the kidney, but failure to find the bacillus does not warrant the conclusion that tuberculosis does not exist. In tuberculosis of the kidney, as in all other forms, there is a certain "cachexia" or general failure of health which is not generally present in renal calculus.

Sarcoma of the kidney may be confounded with renal calculus, yet the symptoms of the former are quite unlike those of stone. Sarcoma is sometimes painless, and the first indication of its presence is a tumor or hæmaturia or both. More commonly in my experience sarcoma is attended by a deep wearing pain, which is pretty constant, is accompanied by tenderness, and by occasional profuse hæmaturia; tumefaction can be detected quite early, and the form of the kidney will be distorted. In one of my cases calculus was excluded by Fenger and myself on the ground that the pain covered too large an area and that the hæmaturia was too profuse. Subsequently I removed a sarcomatous kidney.

Prognosis.—Renal calculi are not sources of immediate danger. Frequently the passing of a single calculus is the beginning and end of the patient's trouble, since a patient who has had one attack of renal colic promptly puts himself under treatment for the purpose of escaping another. Many patients pass calculi at intervals for years and yet remain in good health. Nevertheless, a renal calculus which is not gotten rid of *per vias naturales* becomes the source of great annoyance and pain, and sooner or later demands removal by lumbar nephrolithotomy—an operation which experience has now rendered both safe and successful in the majority of cases.

Treatment.—In many cases the treatment will—or should—be prophylactic rather than curative. In the early stage, when it is a case of "gravel" rather than calculus, the object of the practitioner should be to prevent the formation of a calculus. The treatment will depend

upon the variety of "gravel" present. In a case of lithuria with highly acid urine of high specific gravity, which upon cooling deposits a copious precipitate of "red pepper" crystals, the appropriate treatment will be the exhibition of alkalies freely diluted with pure water. The vegetable acid alkalies are preferable. I prefer the potassium citrate in doses of thirty grains every three or four hours, dissolved in nearly a glass of water. Potassium acetate, sodium acetate, or lithium citrate may be given instead, but I have seen the best results from the use of potassium citrate. The liver usually requires attention. An evening dose of a grain of calomel and five grains of sodium bicarbonate, followed the next morning with half a glass of Rubinat-Condal water, will arouse the hepatic function. The patient should be directed to drink freely of pure water, but he should partake sparingly of meat. This treatment, persisted in for some time, will generally unload the urine of lithic acid and cause the symptoms of calculus to disappear, although they are liable to return again and again for months and even years. Some benefit will be derived from the lithia waters, of which there are several varieties in the market. Some of the so-called lithia waters contain more calcium than lithium, and these should be avoided.

If the patient can pass a "season" at Carlsbad, he will be much benefited thereby; if he is unable to do that, the Carlsbad Sprüdel salt, which can be easily obtained, will be of considerable benefit.

Abstinence from alcoholic and malt liquors and the very temperate use of tobacco—if used at all—should be insisted upon.

If it be a case of oxaluria or cystinuria, a few minute doses of calomel and soda, followed by the Rubinat-Condal water, should be given, after which bitter tonics combined with nitro-hydrochloric acid will be found very useful.

The following is a favorite formula of mine:

℞. Acidi nitro-hydrochlorici diluti,
Tincturæ nucis vomicæ, *āā.* ʒiiss;
Syrupi aurantii corticis, ʒij;
Tincturæ gentianæ compositæ, q. s. ad ʒviij.—M.
Sig. Take a tablespoonful before meals in water.

The "nervous" subjects of oxaluria frequently require tonics addressed especially to the nervous system, such as the elixir of iron, quinine and arsenic, or the compound syrup of hypophosphites. In these cases also the free use of water should be inculcated.

Oxaluric patients frequently require rest, or rather recreation, before any marked improvement takes place.

The treatment of nephralgia due to the transit of a calculus is purely "expectant." The patient must be sent to bed; hot anodyne fomentations must be applied over the seat of pain, and morphine must be given hypodermically, as the case may require, although I have derived quite as good results from administering the remedy per rectum. Half a grain of morphine with a fiftieth of a grain of atropine, dissolved in a couple of drachms of warm water and thrown gently into the rectum, will be found very prompt and efficient in the relief of renal colic. I have also found hot rectal (or rather colonic) enemata very comforting

during the passage of a calculus through the ureter, and I have fancied that it expedited the agonizing journey by relaxing the ureter.

When a calculus becomes too large to escape through the ureter, and is therefore retained in the pelvis of the kidney, the question of treatment soon becomes a serious one. There is no reason for believing that renal calculus can be dissolved and removed by medicinal agents. The pain may be relieved by quietude and anodynes; pyrexia may be lessened and improved by the treatment already suggested for suppurative pyelitis; hæmaturia may be arrested by astringents; and various other symptoms may be combated by appropriate makeshift remedies, but the only radical remedy for nephro-lithiasis is nephro-lithotomy. This operation, first performed by Henry Morris in 1880, has proved very successful in the hands of various surgeons. I have had occasion to perform it 7 times, with 1 death, and I recommend it in all cases where the symptoms of stone are positive and where the age or condition of the patient does not contraindicate it.

HYDRO-NEPHROSIS.

Definition.—When the kidney or its pelvis becomes overdistended by mechanical retention of urine or any other non-purulent fluid it is termed hydro-nephrosis. At different periods it has been called "hydrops renis," "hydrops renalis," "hydro-renal distention," and "cystic tumor of the kidney," but in 1841, Rayer proposed the name "hydro-nephrosis," which has been generally accepted. The term "cystic tumor of the kidney" is entirely inappropriate; besides, it is already in use to designate true cystic degeneration of the kidney.

Etiology.—The causes of hydro-nephrosis fall into two groups—namely, (*a*) congenital and (*b*) acquired causes. By congenital *causes*, however, must not be understood congenital hydro-nephrosis, which is a very different condition and will be considered presently.

(*a*) *Congenital causes* are those which exist before birth, but may not give rise to a palpable hydro-nephrosis until months or even years after birth. Among these causes may be enumerated twists of the ureter, great obliquity of the ureter at its point of origin in the pelvis, valvular flaps or folds which partially close the pelvic orifice of the ureter (as in Fenger's case), contraction of the vesical orifice of the ureter, so that the escape of urine is retarded, or pressure of the renal artery passing in front of instead of behind the ureter.

In one case hydro-nephrosis was produced by "mucus retained in one half of a bifid ureter" (Morris).

In some cases the vesical orifices of the ureters have been found "thick, rigid, and of merely pinhole size" (Reynard). Strange[1] records a case of diabetes insipidus in a farm laborer aged eighteen in whom the sectio cadaveris revealed "complete absence of all proper parenchymatous substance, both tubular and cortical," the kidneys being "reduced to mere sacs of from twice to thrice the extent of the healthy kidneys."

[1] *Beale's Archives*, 1862.

The sacs were divided into a number of lobules by the interlobular connective tissue which remained undeveloped in the fœtal state. Strange was "disposed to view the case as at least in great part congenital, since such an amount of destruction of kidney substance could have scarcely gone on without some evidence of it, either in the urine or in the general health." Mr. Glass records a case in the *Philosophical Transactions* in which thirty gallons of fluid were withdrawn from a hydro-nephrotic cyst, the cause being the tortuous course of the ureter on its way to the bladder. In case of a child five years of age which was brought to my clinic a few years ago I drew nearly two gallons of fluid from the right kidney by aspiration, but I do not know the subsequent history of the patient.

It will readily be seen that while the causes above given are of prenatal origin, their effects may be produced so slowly that months or years may elapse before the kidney becomes distended so as to produce a palpable tumor.

(*b*) *Acquired Causes.*—Chief among acquired causes are cancerous or other tumors of the pelvic organs, enlarged ovaries, chronic cystitis with hypertrophy, vesical calculus, tumors of the bladder (including enlarged prostate), tumors of the abdominal organs compressing the ureter, stricture or contraction of the ureter following injury, twist of the ureter (especially liable to occur in cases of "floating kidney"), and renal calculi partially or completely plugging the ureter. I have seen 2 cases in which a large calculus lying in the ureter was channelled or grooved so as to allow a partial escape of the urine; in both cases there was well marked hydro-nephrosis. Any cause which slowly or partially obstructs the ureter may result in hydro-nephrosis; if the obstacle exists in the renal pelvis, the calibre of the ureter may not be essentially altered, but if the obstruction is located in the bladder, the ureter is invariably dilated, elongated, and thickened so as to resemble coils of intestine. Morris calls attention to the fact that frequent micturition aids materially in the production of hydro-nephrosis—a statement which I can confirm from my own observation. A long existing and progressive stricture of the urethra is also quite capable of inducing dilatation of the kidney, on account of the thickening of the bladder and the consequent frequent and difficult urination. Then the causes of hydro-nephrosis are entirely mechanical and lie outside the kidney itself.

Course.—The renal pelvis is first involved; it gradually becomes distended into a spherical sac, and the ureter takes part slowly but surely in the process of distention, and ultimately it is likely to be very much enlarged and tortuous, resembling a coil of intestine. Meantime, the calyces are pushed apart, the papillæ are crowded back, and then, in process of time the medullary and cortical portions of the organ are expanded and compressed—and probably in part absorbed—until nothing is left but a rounded and indistinctly lobulated tumor, whereof about one half is represented by the dilated yet somewhat thickened pelvis renalis, while the other half is made up of the dilated and attenuated parenchyma of the kidney. All this is accomplished by the slowly accumulating urine, due to the obstruction, whatsoever and wheresoever it may be. Of course the more perfect the obstruction the more rapidly the

distention takes place, and of course the more rapidly the tumor develops. Usually the pelvis thickens somewhat as the distention proceeds, but sometimes it yields to the pressure and becomes remarkably thin and translucent. In very anæmic subjects the pelvic portion of the hydro-nephrotic tumor is almost certain to be thin and delicate. The size of the tumor varies greatly in different cases. In some rare cases the kidney is scarcely enlarged at all. I have seen one such case. In others it may be moderately enlarged and rounded in form. In still others it may be immensely distended, so as to be in danger of bursting. In such cases the whole abdomen is distended, the diaphragm is crowded up, the heart displaced, the intestines compressed, and the case is likely to be taken for an ovarian cyst. I have known two cases in which a hydro-nephrotic kidney was removed under the supposition that it was an ovarian cyst.

The contents of hydro-nephroses vary. In one case the fluid may be pale and watery, almost clear, of very low specific gravity, neutral in reaction, non-albuminous, and containing no appreciable amount of uric acid or its compounds, but *always* containing sodic chloride. This is likely to happen where the renal substance has suffered practical destruction. In another case the contained fluid will resemble normal urine, except that it will be deficient in urea and of low specific gravity. But nearly all the normal factors of urine may be found, only in less than normal amount. Rarely, a case occurs in which the contained fluid consists of pus or muco-pus, and mortar-like deposits or masses of urinary salts; but all this is due to secondary infection, and the case then becomes one of suppurating pyelitis instead of hydro-nephrosis.

Symptoms.—The symptoms vary greatly in different cases. Unless the distention is sufficient to give rise to a tumor, there may be an entire absence of characteristic symptoms, and yet hydro-nephrosis may be present and of long standing. Moreover, such symptoms as are present may be referable entirely to the cause of hydro-nephrosis rather than to the disease itself—as, for example, when hydro-nephrosis is the consequence of the obstructive pressure of a uterine or ovarian tumor on the ureter. In many cases, however, a palpable tumor in the situation of the kidney will be present; this tumor will be indistinctly lobulated, fluctuating, generally somewhat tender, (although less so than a tumor from pyo-nephrosis), and quite movable, although the range of motion is limited, and of course dull on percussion. When grasped with one hand on the lumbar region and the other in front, a distinctly elastic, "watery" feeling will be elicited, which I regard as quite characteristic. The colon is usually in front of the tumor, but that is not an inflexible rule, and the small intestine may be in front, although it is quite as likely to be thrust to the opposite side of the abdomen, especially if the case be well advanced. If the kidney is very much distended, it may displace the abdominal viscera, crowd up the diaphragm, and interfere with respiration and the heart's action. By its pressure on the colon it will always produce constipation. The position of the tumor is characteristic; it is always in the flank or renal region, causing the parietes of that area to bulge out posteriorly and laterally, especially when the patient is lying on his back slightly inclined toward the affected side.

Of course in cases where there is no essential enlargement of the kidney these symptoms will be wanting.

When both ureters are occluded, as in uterine cancer or villous cancer of the bladder, there will be bilateral hydro-nephrosis. Such cases are rare, yet every clinician of experience has seen them. They are usually complicated by uræmia and other symptoms of anuria, but death usually occurs before the kidneys are very much enlarged.

While it is generally true that hydro-nephrosis is not a painful disease, there are cases in which the distention is so great that the pain amounts to agony. Morris relates a very striking case of this nature, and I have recently seen one in which the pain was very great, yet the kidney was but moderately enlarged. Cases which are caused by renal calculus have a history of paroxysmal pain from the first, but as a clinical fact hydro-nephrosis is rarely produced by calculus, pyo-nephrosis being a much more likely result.

Sometimes nausea, vomiting, anorexia, flatulence, and palpitation or irregularity of the heart accompany hydro-nephrosis, but these symptoms are quite as likely to be due to the morbid antecedent of hydro-nephrosis as to the latter disease itself.

DIAGNOSIS.—The differential diagnosis of hydro-nephrosis ought not to be difficult after sufficient distention has taken place to produce a palpable tumor. But in the early stage of hydronephrotic distention a positive diagnosis may not only be difficult, but impossible. Hydro-nephrosis may possibly be confounded with pyo-nephrosis, but the history of the latter, together with the more dense and less distinctly fluctuating character of the tumor, the persistent pain and tenderness, the purulent and often bloody urine, the daily rise of temperature, with rigors more or less strongly marked, form a group of symptoms so utterly unlike those of hydro-nephrosis that the distinction ought to be easily made.

Perinephric abscess or perinephric extravasation can hardly be mistaken for hydro-nephrosis, presenting as it does an irregular, asymmetrical swelling in the lumbar and lateral region, but not in front; tenderness, swelling, fluctuation, and pyrexia are prompt in their appearance and character, and these symptoms are quite unlike hydro-nephrosis.

Hydatid disease of the liver, spleen, or kidney might be mistaken for hydro-nephrosis, but need not be. Hydatids are always of slow growth; usually painless and without fever; non-fluctuating, but presenting a peculiar elastic tumor, with the characteristic thrill or "fremitus" which cannot be mistaken for anything else; and the hydatid tumor, if splenic or hepatic, can be traced by a distinct connection with the spleen or liver. In a doubtful case aspiration might settle the question by the withdrawal of the hooklets of echinococci along with the fluid. A very large hydro-nephrotic distention may easily be confounded with an ovarian tumor; in fact, many a hydro-nephrotic kidney has been removed under the supposition that it was an ovarian tumor. I have seen two such cases. And yet this error is easily avoided with proper precaution. An ovarian tumor develops from below upward, a renal tumor from above downward; an ovarian cyst is first discovered in the hypogastric region near the median line,

a renal cyst in the lumbar region far away from the median line; an ovarian cyst is closely adjacent to, and frequently displaces the uterus to the right or left, a renal cyst is remote from the uterus and does not encroach upon it; an ovarian cyst may be grasped between the finger in the vagina and the hand over the lower abdomen toward the median line, a renal cyst will be found in the lumbar region, projecting laterally and posteriorly. If aspiration is resorted to, the fluid from a distended kidney will probably have a urinous odor, and saline elements may be detected by the proper reagents, while the fluid from an ovarian tumor will manifest the chemic and microscopic characteristics thereof.

Hydro-nephrosis with extreme dilatation and attenuation of the cyst wall may so closely resemble ascites that the distinction may be difficult. But if the patient is placed upon his back with the knees drawn up, it will be seen that the tumor assumes an outline quite unlike ascites, in that it projects forward and is clearly outlined by a restraining wall. Moreover, aspiration will assist very essentially in correcting the error, since the fluid will contain urinary factors if the case be one of hydro-nephrosis.

It should be remembered that in nearly all renal tumors the large intestine is in front of the tumor: if this fact is kept in mind, it will go a long way toward starting the practitioner in the right track; yet I have lately seen a very large hydro-nephrotic tumor which was in front of the colon.

Prognosis.—Hydro-nephrosis is not necessarily a dangerous disease. If only one kidney is involved, the other kidney is abundantly capable of taking on sufficient extra work to avoid all danger from suppression, and if the disease is slow in its progress, the danger, if any, will be very remote. If both kidneys are implicated, the prognosis is of course very grave, the danger being more or less imminent as the progress of the disease is more or less rapid. Yet life is sometimes prolonged until both kidneys are apparently almost totally destroyed; but, as the destructive process is slow, other organs gradually acquire the power of becoming in some degree "vicarious" kidneys, and the system learns to tolerate the urinary factors without serious complaint.

In hydro-nephrosis immediate danger is frequently greater from the cause than from the disease in question. In most cases, even of bilateral hydro-nephrosis, life may be greatly prolonged by the resources of surgery, and in unilateral cases radical cures can in most instances be effected. No general prognostic rule can be laid down: each case must be regarded as *sui generis*, and its future must be predicted after studying its peculiar causes and environment.

Treatment.—Of course no internal medication will be of the slightest use so far as the cure of hydro-nephrosis is concerned. Symptoms or complications resulting from hydro-nephrosis may require treatment, in which case the practitioner must adapt the remedy to the indication. But the treatment of hydro-nephrosis, *per se*, is purely mechanical or surgical.

Manipulation or friction is said to have resulted in cures; Roberts and Broadbent each report a case so cured. It is probable that the manipulation untwisted or straightened the ureter or pushed along an impacted calculus, or in some way dislodged or removed some impedi-

ment to the flow of urine which was in its nature transitory, and hence a permanent cure resulted. But such cases are few and far between. In cases of extreme distention manipulation or massage would be positively dangerous, since rupture of the thin-walled tumor might result, and under any circumstances manipulation must be practised with great care and gentleness.

Aspiration is a safe and legitimate method of treating hydro-nephrosis. Several permanent and radical cures have resulted from aspiration. In some instances the operation had to be repeated several times. Not infrequently, however, it fails as a means of cure, but produces marked temporary relief, and, as the operation is practically without danger when properly performed, it should not be denied in well selected cases.

If aspiration fail, the only remaining resort which amounts to anything is nephrotomy with free opening of the cyst, stitching the border of the cyst wound to the free border of the wound in the parietes. This is a simple and comparatively safe operation, and generally results in cure. A urinary fistula may remain for a time, but it will generally close up in from six to twelve months.

The reader is referred to the works on surgery for detailed directions as to the performance of aspiration and nephrotomy.

RENAL TUMORS.

TRUE NEOPLASMS of the kidney of sufficient size to permit of recognition during life are not common, but small nodular growths are by no means rare, as the post-mortem table is constantly demonstrating. Time and again have I found post-mortem explanation of renal pain and hemorrhage in a small tumor in the kidney whose existence was unsuspected or at least merely conjectured.

The neoplasms most likely to occur in the kidney are the following:

1. Non-malignant:
   - (*a*) Fibromata,
   - (*b*) Lipomata,
   - (*c*) Angiomata,
   - (*d*) Adenomata,
   - (*e*) Papillomata.
2. Malignant:
   - (*a*) Sarcomata,
   - (*b*) Carcinomata.

1. NON-MALIGNANT TUMORS.

(*a*) **Fibromata.**—In the great majority of cases these growths are of no clinical importance. They are generally minute nodules of connective tissue, producing no symptoms, totally unrecognizable during life, and practically harmless as to results. A few cases are on record, however, in which fibrous tumors attained considerable size and re-

quired surgical interference. They are not always simple fibromata, but may contain "two or more distinct histological elements" (Newman), as in Billroth's case of fibromyoma. This fact, however, does not essentially alter the clinical history of the tumor or render it more or less amenable to treatment. Fibrous tumors of the kidney are rather more likely to undergo fatty, calcareous, or cystic degeneration than in other localities.

(*b*) **Lipomata** occur very rarely in the kidney. I have never met with a case. Newman, who has investigated the subject of renal tumors exhaustively, is not aware of a single case of fatty tumor of the kidney which "called for surgical interference." Circumrenal fatty tumors are sometimes found, Sir Spencer Wells having removed two, weighing sixteen and a half and fourteen and a half pounds respectively.

(*c*) **Angiomata** are equally rare. They are generally located in the pelvis or at the surface of the kidney immediately beneath the capsule, in the latter place having probably been developed from the stellate vessels. In the pelvis they usually induce hemorrhage (hæmaturia), sometimes in large amount.

A differential diagnosis is impossible, although recurrent attacks of profuse hemorrhage without the lancinating pain of renal calculus ought to arouse the suspicion of some variety of vascular growth in the pelvis of the kidney, unless the symptoms point distinctly to the bladder as the seat of trouble.

(*d*) **Adenomata** are not very rare, but they are of very little clinical consequence, since they rarely secure recognition or demand treatment. Newman has recorded only three cases which required surgical attention. They are scarcely ever larger than a bean, and may be of only microscopic size. They have no special interest for the physician and almost none for the surgeon.

(*e*) **Papillomata** are equally rare, being scarcely ever met with in the kidney; hence they have very little clinical or pathological importance. The causes which lead to the production of papillomata are of very feeble potency in the kidney; hence the results are correspondingly barren.

Symptoms.—The symptoms produced by the foregoing tumors are few in number and feeble in expression, except in those rare cases where sizable tumors are produced. There may be some slight pain, but not sufficient to impress the patient or cause him to complain. No tumor or enlargement can be detected, and there is no tenderness on pressure. In cases of angiomata or papillomata there may be slight hæmaturia, and, in fact, it may be quite persistent and wholly inexplicable, at least beyond the conjectural stage. But where there are frequent attacks of hæmaturia which cannot be attributed to cystic disease, and at the same time slight transitory pain in the kidney, the practitioner has pretty good reason for suspecting the presence of a non-malignant tumor, even though he cannot recognize it by persistent palpation.

In cases of fibromata of large size symptoms are not wanting. There is a palpable nodulated tumor, dense, hard, elastic, and somewhat tender, occupying the place of the kidney. Its form suggests an enlarged kidney. There will generally be a history of slight occasional hæmaturia, especially after unusual muscular effort, and the hemorrhage is sometimes quite profuse.

There will be a history of repeated attacks of dull pain, but no sharp or lancinating pain. These symptoms point pretty clearly to renal fibroma, although other renal tumors might possibly occasion precisely similar symptoms.

2. Malignant Tumors.

(*a*) **Sarcomata.**—Newman recognizes seven varieties of renal sarcoma, but this is a very needless refinement. Clinically, renal sarcoma is almost invariably of the "mixed" variety—that is, a mixture of "round" and "spindle" celled sarcoma, the former predominating if the growth be rapid, and the latter if it be slow. Sarcoma is peculiarly a disease of early life, the average age in 37 cases collected by Windle being ten years and two months. It may occur in adult life or even in old age, but very rarely. With almost equal rarity sarcoma may attack the fœtus in utero, the variety then being "myo-sarcoma," the muscular element being due to faulty "segmentation of the formative muscular plates, whereby some of the cells become included in the rudimentary kidney" (Cohnheim). Congenital sarcoma may attack one or both kidneys; it may reach enormous dimensions even before birth, so as to render natural delivery impossible, or it may grow with great rapidity after birth, until the whole abdomen seems to be occupied by it.

Congenital sarcoma of the kidney is usually fatal within a year after birth, but this is not an invariable rule. I have seen 2 cases of renal sarcoma in children of three and four years of age respectively, 1 of which occupied the entire abdominal cavity, and indeed all the abdominal organs, in its predatory growth. The abdomen was enormously distended, and the little patient was emaciated to the last degree. The other case (a girl of four years) involved only one kidney, which was enormously enlarged. Nephrectomy was successfully performed by J. E. Owens, but the disease attacked the remaining kidney within twelve months after the operation.

Primary sarcoma of the kidney is rare, and is generally regarded as congenital. I have seen 2 cases of primary renal sarcoma, 1 case being a girl aged eighteen, the other a man aged forty-three. In both these cases I performed nephrectomy with favorable results, but in the case of the girl the disease reappeared in the remaining kidney two years after the operation.

The duration of renal sarcoma varies greatly. It depends largely upon the form or type. Round celled sarcoma progresses rapidly, is rapidly disseminated, and is therefore rapidly fatal; spindle celled sarcoma grows slowly, is generally local, and therefore is less rapid in its results. It is a singular fact that "mixed" sarcoma—round and spindle celled combined—is generally more rapidly fatal than round celled alone. Of course the absolute duration in any given case cannot be accurately fixed, since we must calculate from the time it was first discovered.

Symptoms.—There are no symptoms especially characteristic of renal sarcoma, yet there are certain symptoms which strongly indicate this disease. I mention these in order of their occurrence:

First, pain of a deep, dull, wearing type, not sharp and cutting, like

the pain of renal calculus. It is somewhat constant rather than paroxysmal, and is generally increased by violent exercise, like running or jumping. It is quite definitely localized, and does not produce retraction of or pain in the testicles, as is so common in cases of renal calculus.

Secondly, hemorrhage, which generally comes on suddenly and unexpectedly, is quite copious, and is unattended by the torturing, atrocious paroxysmal pain of renal stone. After the first attack of hæmaturia other attacks are likely to follow in quick succession, so that it becomes a prominent symptom in the subsequent history of the case. While the quantity of blood lost in different cases varies greatly, it is in my experience always greater than occurs in connection with renal calculus, and thus becomes an important differential factor.

Third, the appearance of a tumor. In clinical experience the tumefaction produced by a renal sarcoma is not discovered until a considerable time after the foregoing symptoms—pain and hemorrhage—have been present. The character of the tumor is quite suggestive; it is uniform, with here and there a dense rounded nodule projecting from its periphery. It is non-fluctuating, dense, somewhat elastic, and its surface is smooth, except for the large rounded projections already mentioned. It is tender when firmly compressed between the hands, but it is not the acute cutting tenderness of a kidney containing a calculus or the peculiar "sore" tenderness of a suppurating kidney.

Examination of the urine rarely sheds any light on the renal tumors. It is a well known clinical fact that the elements of renal tumors are almost never discharged with the urine, and in those exceptional cases when it does occur it is too late to be of any practical service.

Renal sarcoma may be mistaken for renal calculus or carcinoma. I have endeavored to point out the distinctions between sarcoma and calculus in the foregoing pages; the points of difference between sarcoma and carcinoma will be enumerated presently.

Prognosis.—The prognosis of renal sarcoma is always unfavorable. A few cases progress slowly, so that the patient may survive for several years; but most cases, and especially those which occur in children, progress with great rapidity, and cause death in from six to twelve months after they are first discovered.

Treatment.—The treatment is entirely surgical, except those palliative measures which special symptoms demand. Pain must be relieved by opiates, and my preference is for opium pure and simple. It may be combined with astringents, as gallic or tannic acid, if there be hemorrhage. The opiate should only be given when required, but then it should be employed in full doses, so as to produce a pronounced effect, and this remark applies especially to hemorrhagic cases, crude opium being a most efficient hæmostatic. I have lately employed fluid extract of *Eucalyptus rostrata*, or "red gum," in hæmaturia with excellent effect; if pain be present, it may be combined with the deodorized tincture of opium. The preparations of iron and other tonics will be required; the bowels should be kept studiously regular, as the pressure of a loaded colon on the affected kidney must be avoided. The patient should be enjoined to keep quiet, and especially to avoid all active or violent exercise.

The question of nephrectomy is one of great gravity, and must be decided after weighing all the *pros* and *cons* in each individual case. My own experience in performing nephrectomy for renal sarcoma is limited to two cases: in one the disease invaded the remaining kidney two years after the operation, and the patient died six months after the second invasion; in the other the patient is now (five years after the operation) in excellent health, with no symptoms of a recurrence.

(*b*) **Carcinomata.**—Carcinoma of the kidney appears mostly under two varieties, encephaloid and scirrhous, whereof the former is by far the most common. Colloid and epithelioma have been found in the kidney, but so rarely as to be of little clinical import.

Primary cancer of the kidney was formerly believed to be all but unknown, but we now know that, although it is quite rare, every clinician of much experience has met with cases which were indubitably primary. Secondary cancer of the kidney is by no means uncommon, although, as Newman suggests, it is probable that many cases of sarcoma are classed as carcinoma—a mistake which ought not to be made at the present day. Primary cancer is generally limited to one kidney, but may invade the remaining kidney as it progresses. Secondary cancer is very likely to attack both kidneys at the same time, since the same source of infection—the blood—reaches both kidneys with equal freedom.

Renal cancer is by far more common in childhood than in adult life, Newman's table stating that 48 per cent. of the cases occur before the age of ten years. These cases are almost invariably encephaloid, and they sometimes attain enormous size, in one instance, quoted by Newman, weighing thirty-one pounds. Specimens weighing four or five pounds are not at all uncommon.

Symptoms.—Roughly speaking, the symptoms of renal cancer are the symptoms of renal sarcoma over again. So nearly are they alike that it is sometimes impossible to differentiate them; yet a careful observer will distinguish certain points of difference which will enable him to make a fairly accurate "working" diagnosis of renal cancer.

First, the pain of renal cancer is of a more pronounced and lancinating character than the pain of sarcoma. It comes on without apparent cause while the patient is at rest or in bed, while the pain of sarcoma follows exercise or muscular effort. This distinction vanishes in the later stages of the latter, but it is a valuable differential factor in the early history of a case. Again, the pain of cancer is more radiating or "reflex," especially travelling along the splanchnic tract, somewhat like the pain attending renal calculus, only less acute, while the pain of sarcoma is more localized and fixed, so that the patient can point out its exact locality. Lastly, the pain of carcinoma has a persistent wearing character, unlike the more tractable pain of sarcoma.

Secondly, hemorrhage is about equally prominent as a symptom in cancer and sarcoma, but there is always a possibility that the hemorrhage of cancer will be attended by escape of shreds of breaking-down tissues, which can be secured for microscopic examination, and that the results of the examination will be strong confirmation of the diagnosis. But hemorrhage from sarcoma, except in its late stages, is almost never attended by the discharge of tissue débris.

Third, tenderness is developed at an earlier period, and is much more strongly marked in cancer than in sarcoma. The patient shrinks and complains of being "hurt," while in sarcoma the tenderness is present, but is of the endurable kind, which the patient does not contemplate with fear and apprehension. After two or three examinations of the same patient the practitioner will see the practical force of this point, and especially the dread with which the patient, generally a child, submits to a manual examination, and the manifest evidence which he gives of being "hurt" by the manipulations; but this is manifested in very much less degree in sarcoma.

Fourth, the tumor of renal cancer is quite different from that of renal sarcoma. It is less symmetrical, less "reniform," is more likely to be nodulated and irregular or to present larger projecting masses than sarcoma, which generally maintains a pretty well defined "kidney shape." The tumor is elastic, not fluctuating, yet it has a semi-fluctuating, yielding character under bimanual pressure, unlike the more dense and less elastic quality of sarcoma. Moreover, the cancerous tumor enlarges much more rapidly than the sarcomatous, and as it grows its ugly form becomes more and more apparent.

Of course I am now speaking of encephaloid cancer of the kidney, since scirrhus rarely presents any tumor at all, although the pain and tenderness will be persistently present.

It remains to speak of the so-called cancerous "cachexia"—the sallow complexion, the failing health, the emaciation, the drawn and wrinkled face so expressive of suffering, and so strongly pathognomonic of cancer; which, added to the symptoms already detailed, make up a picture which can hardly be mistaken for sarcoma or anything else if the practitioner is careful and methodical in his analysis of the case.

Treatment.—There is nothing to be done in the way of cure unless it be extirpation of the kidney involved, and even that does not guarantee a permanent cure, since experience shows that the disease is likely to attack the remaining kidney in from four to five years. Nevertheless, it does prolong life, and should be performed in proper cases. But if other parts or organs are involved, or if the diseased kidney is very greatly enlarged, or if the general health of the patient is very much impaired, nephrectomy promises nothing and should be avoided.

The efforts of the physician must necessarily be restricted to the relief of pain, hemorrhage, and other complicating symptoms, and the reader is referred to the remarks on the Treatment of Sarcoma (page 792), all of which are entirely applicable to the treatment of renal carcinoma.

Cystic Degeneration of the Kidney.

Definition.—A general cystic metamorphosis of the renal tubules and Malphigian bodies, with corresponding disappearance of the renal parenchyma; as a rule, both kidneys are involved, although one may be in a more advanced state of degeneration than its fellow.

Pathological Anatomy.—The kidney is enlarged, sometimes enormously. In Hare's case—so often quoted—the left kidney weighed sixteen pounds, but this is an extreme case. It is not unusual to find the cystic kidneys weighing two or three pounds. They generally re-

tain the form of the kidney more or less perfectly. But the whole organ is converted into a mass of cysts which vary in size from a pin's head to a pigeon's egg. These cysts are lined with a thin, diaphanous membrane upon which rests an irregular layer of rounded epithelial cells, and they are filled with fluid which may be pale or yellow or dark brown in color—may be thin or viscid or syrupy in consistence, and frequently contain abundant crystals of cholesterin and numerous degenerated epithelial cells. Microscopic examination of sections will reveal the presence of isolated patches of renal structure not yet involved in the general ruin, and sections of tubules may frequently be traced to the point where they are suddenly dilated into minute cysts. David Newman has clearly shown that the cysts are the product of dilated tubules and Mal-

FIG. 52.

Case of cystic degeneration: left kidney, exterior.

phigan bodies—in fact, a congeries of true retention cysts. The accompanying photogravures, taken from a very perfect specimen of cystic degeneration of the kidney—show the appearance of the organ very clearly. The same patient's right kidney was almost as far advanced

in cystic degeneration, and it is generally the case that both kidneys are involved, although one is apt to be more damaged than the other.

Similar cysts sometimes coexist in the liver and spleen, "though so infrequently that it may be doubted whether the connection is more

FIG. 53.

Case of cystic degeneration: left kidney, longitudinal section.

than accidental" (Howship Dickinson). There is generally hypertrophy of the heart, as in renal cirrhosis.

ETIOLOGY.—No definite cause can be assigned for this disease. It is more common in males than in females; it always occurs in adults, and must not be confounded with congenital cystic degeneration, which is generally fatal before birth.

W. H. Dickinson thinks "it is possible that in some cases the cystic change may be due to obstruction by tubal nephritis, yet, as a rule, it is due to a form, perhaps a peculiar form, of interstitial fibrosis."

SYMPTOMS.—There are no special symptoms which indicate the presence of this singular disease. In many cases it has first been discovered by post-mortem examination.

So far as they go, the early symptoms are practically those of interstitial nephritis. There is arterial tension, a sharp, angry systolic sound, a somewhat rapid pulse, with an abundance of pale urine, which is of low specific gravity, turbid with mucus, slightly albuminuric, and contains scattering leucocytes. At a later stage the swollen kidneys may

be recognized upon examination, and the projecting cysts may be felt in thin patients. At this stage the urine occasionally contains blood globules, the albumin will be increased, leucocytes and degenerated epithelia will be abundant, and plates of cholesterin are apt to be present. Suppression of urine is liable to occur with coma or convulsions, or both, and death soon follows. But years may elapse between the early symptoms—which may be so feebly marked as not to attract attention at all—and the later symptoms which denote the extreme gravity of the patient's condition. In some cases there is pain in the loins, even in the early stage, but this is the exception rather than the rule.

Diagnosis.—It is rarely the case that a diagnosis is made in the early stage of cystic disease of the kidney. It is certainly all but impossible to differentiate cystic disease from renal cirrhosis at this period. In the few cases which are attended by pain in the loins of a dull, deep aching kind, not severe, but quite persistent, cystic disease may be suspected, inasmuch as interstitial nephritis is not attended by local pain. At a later period the swollen kidneys, the urine turbid with fatty epithelia and cholesterin, together with occasional hæmaturia, would furnish excellent grounds for a positive diagnosis.

The sallow aspect of the patient, the progressive anæmia, and the gradual failure of vigor and vitality would also help in establishing the diagnosis of cystic disease.

Prognosis.—The disease is invariably fatal. Many cases run a very chronic course, and it is wonderful how long patients will survive after the kidneys are apparently absolutely destroyed.

The case from which the preceding figures (pp. 795, 796) were taken illustrates this point. Both kidneys seemed to be entirely ruined, yet the patient kept about her work—that of a missionary—until within four weeks of her death. Some cases seem to have latent periods of long duration; then suddenly the process of destruction is resumed with increased vigor. But every case tends toward, and sooner or later reaches, a fatal termination.

Treatment.—No treatment, medical or surgical, is of any avail. Elimination must be helped along by other channels than the kidneys. The bowels must be kept free and the skin should be made to do its work. Tonics will be needed on account of the inevitable anæmia.

Various symptoms may arise which must be met and treated according to the indications, but nothing can be done to stay the progress of the disease or rescue the patient from its consequences.

RENAL ABSCESS.

By renal abscess we do not mean any of the infective forms of suppuration characterized by a multitude of minute collections of pus scattered through the kidney, or that form of suppurative disease known as "pyelitis" or "pyo-nephrosis" (already described), but rather a circumscribed collection of pus of sufficient size to merit the name "abscess,"

situated in the parenchyma of the kidney and environed by a well defined wall of limitation; in other words, an abscess of the kidney has the same physical and structural features as an abscess in any other location.

Etiology.—There are several causes of renal abscess. Perhaps the most common are traumatic causes, as blows, kicks, or similar injuries due to violence. The immediate result will be contusion, followed by infection and suppuration. In such cases it will not always be possible to trace the source or route of the infection, but the fact remains.

It must not, however, be inferred that every injured kidney undergoes suppuration, although the liability is always present. In wounds of any kind involving the kidney the danger of suppuration amounts to almost a certainty, but the suppurative process may not assume the form of an abscess.

Calculi impacted or encysted in the parenchyma of the kidney are liable to provoke circumscribed suppuration, and this likelihood is very greatly increased by violent exercise or sudden "shocks" caused by falling or jumping.

Another source of renal abscess is the coalescence of a number of small purulent foci, especially if they be tubercular. The tissues surrounding an abscess cavity are always liable to invasion by the restless leucocytes which pervade the neighborhood; and, while the "phagocyte" theory is certainly interesting and probably true, it is yet certain that suppuration and tissue destruction follow invasion; hence it is that the barriers which separate contiguous pus cavities in the kidney are broken down, and in place of a dozen little abscesses we have one large one, with firm resisting walls which restrain the leucocytes from further depredations. The size of such an abscess will depend very much upon the number of minute cavities contributing to its dimensions.

Again, a renal abscess may be due to an embolic infarction, the plugs being conveyed from the heart directly (as in ulcerative endocarditis) or from some point along the aorta between the heart and the origin of the renal artery, as in aortic aneurysm. In such cases the embolism is followed by an area of wedge-shaped or pear-shaped infarction, which sooner or later becomes infected, suppuration follows, and a pear-shaped abscess is the final result. If the arrest of circulation is due to ulcerative endocarditis, the infective micro-organisms are brought along by the emboli, since ulcerative endocarditis is itself infective. Such an abscess is all but sure to involve at least one entire Malpighian pyramid, and may involve the entire kidney.

Renal parasites sometimes provoke suppuration sufficiently grave to merit the title "abscess."

Irritant drugs, like copaiba, turpentine, and cantharides, may cause renal abscess. I have recently seen a case of renal abscess which terminated fatally produced by the treatment pursued by a charlatan for the cure of "lost manhood." The patient, a young man of twenty-two, was a splendid specimen of physical perfection until he commenced the course of "treatment" which led to his needless death.

"Metastatic" abscesses are as likely to occur in the kidney as in the liver; they are generally multiple in the beginning, but may coalesce in the manner already described.

PATHOLOGICAL ANATOMY.—Renal abscess is generally unilateral. In a typical case there is but one abscess, which is well defined and accurately circumscribed, but sometimes we find several separate pus pockets in the same kidney. More frequently several small abscesses have coalesced, forming an irregular cavity with "compartments" which indicate the locality of each of the several original abscesses. After a time the abscess is likely to form communication with the pelvis of the kidney, and thence through the ureter into the bladder, or it will open through the capsule into the perirenal tissues, with the symptoms of perirenal added to those of renal abscess. If the abscess opens into the pelvis, partial drainage through the ureter and bladder will follow, and it may happen that a renal abscess will result in complete recovery after being spontaneously drained through the ureter. Sometimes the contents of an abscess are partly absorbed, partly encysted, and the putty-like remains continue harmless during life.

SYMPTOMS.—There are no pathognomonic symptoms of renal abscess. The symptoms frequently resemble those of pyonephrosis, and, as Morris remarks, many of the cases reported as renal abscess *are* cases of pyonephrosis.

In an acute case the first symptoms will be pain in the diseased organ, of a dull, throbbing character; not the intense, cutting pain of stone. Along with pain, or following it after a brief interval, will be fever with rigors, rapid pulse, coated tongue, and anorexia. If the abscess be caused by injury, hæmaturia is likely to precede all other symptoms; if it is metastatic, there will be no hæmaturia.

The affected kidney rapidly becomes tender, and the tenderness is of the sharp, pronounced kind which indicates an acute process. The kidney is rarely much enlarged, the pus escaping before any palpable tumor forms. In some cases of great acuteness and violence the affected organ can be felt by bimanual palpation, and well marked fluctuation can be detected. Much diagnostic aid can generally be derived from the history of the case, traumatism, infection, or both, being almost invariably present as an antecedent.

Sometimes we meet with a subacute or chronic abscess of the kidney which forms without any marked symptoms, and is first discovered post-mortem. But such cases are usually accompanied by general loss of health, slight evening pyrexia with perhaps rigors, and later on by pyuria, so that the careful diagnostician can find guiding symptoms by persistently looking for them.

TREATMENT.—If the physician suspects from the history of the case that an abscess is forming, the patient should be sent to bed and kept absolutely quiet; leeches should be applied, followed by an ice bag or a cold water coil, and anodynes, the best of which is opium, should be freely given. The diet should be liquid, and restricted as to quantity. If, in spite of this treatment, the abscess seems progressing, hot applications should be substituted for the cold, and the physician should watch carefully for symptoms of tenderness and fluctuation. As soon as it becomes apparent that a pus collection has formed free incision through the loin should be resorted to and effectual drainage should be established. An exploratory incision would be perfectly justifiable in doubtful cases where the weight of evidence was in favor of suppuration.

The sooner the pus can be reached and evacuated the less will be the disturbance of renal parenchyma, which is a very important consideration. Daily antiseptic irrigation will be required and the drainage must be absolutely free and unobstructed.

During convalescence quinine, iron, strychnine, and good diet will be in order, and the question of renal and hepatic elimination must not be forgotten.

PERIRENAL ABSCESS.

DEFINITION.—Perirenal abscess is the result of suppuration of the connective and adipose tissue forming the "fatty capsule" of the kidney. It is not a very common affection, but is occasionally met with both in hospital and private practice.

Three forms are recognized:

(*a*) Primary perirenal abscess—cases which occur independently of any cause traceable to the kidney.

(*b*) Consecutive perirenal abscess—cases in which the disease follows inflammation of the kidney, but in which there is no fistulous opening or urinary infiltration.

(*c*) Consecutive perirenal abscess—caused by escape of pus or urine, or both, through a fistulous opening connecting with a diseased kidney.

ETIOLOGY.—(*a*) Primary perirenal abscess is generally due to some form of injury, such as blows, strains, kicks, and the like. A sudden and unexpected muscular strain, such as occurs in a desperate attempt to avoid falling on the ice, has been known to produce perirenal abscess. Dickinson relates a case caused by "rolling and tossing during a stormy passage from Queensland to England, the whole left side being extensively ecchymosed." In six of Poland's cases no cause could be assigned.

(*b*) Consecutive or secondary perirenal abscess is due to some cause transmitted from the kidney. It may be a severe infective acute nephritis, such as follows scarlatina or diphtheria, in which it is altogether probable the infective microbes make their way through the capsule of the kidney into the perirenal tissue. But a much more frequent cause is the direct transmission of infective material through a renal fistula caused by tubercle, calculus, or suppurative pyelitis. In such cases, of course, the cause is not far to seek, and the perirenal suppuration simply becomes a complication which if promptly recognized and properly treated does not add materially to the gravity of the case. I have seen one case in which perirenal abscess was due to the escape of a renal calculus from the cortex of the kidney through a fistulous opening into the "fatty capsule;" in this case no calculus or other renal disease was suspected, although it occurred in the practice of an excellent and experienced diagnostician. It emphasizes the statement I have already made, that a calculus may remain for years in the renal parenchyma practically without pronounced symptoms. Morris states that "perinephritis is often the consequence of inflammation from distant parts, such as the pelvis, colon, testicle, spermatic cord, spleen, liver, gall

bladder, or vertebræ. So easily can inflammation which begins in the pelvic or iliac regions spread upward to the loins, to the veins along the retroperitoneal connective tissue, and then give rise to an abscess around the kidney, that diseases of, or operation upon, the rectum, urinary bladder, testicle, or the uterus, and cellulitis arising during the puerperal state, are well known causes." For more than twenty years I have been performing frequent operations upon the pelvic and genito-urinary organs, but have never encountered a perinephric abscess as a consequence.

SYMPTOMS.—Constitutional generally precede local symptoms. They are such as suppuration excites elsewhere—rigors, followed by high but vacillating temperature, diaphoresis, dry coated tongue, thirst, anorexia, and sometimes delirium. These symptoms are most marked when perirenal abscess follows some acute septic fever, as scarlatina, small-pox, or diphtheria, but they are always present in some degree. Pyrexia may be distinctly intermittent, so as to closely simulate that of intermittent fever—a fact which the physician must bear in mind lest he be led astray. Constipation of a most obstinate nature is always present, and the muscular effort attending defecation is dreaded and avoided by the patient on account of the pain it produces. So common and constant is this that it becomes an important diagnostic point.

When perinephritis follows some other suppurative process, like pyelitis or cystitis, its symptoms may be completely overshadowed by the pre-existing disease, which has become so prominent in the mind of the physician as to obscure everything else. But the occasion of rigors, sweating, and fever, and especially the constipation and pain at stool, should direct attention to the possibility of a perirenal abscess.

The local symptoms sometimes rapidly follow those already described; sometimes they are long delayed and are not well defined. The first local indication of perirenal abscess is pain. It is always deep seated, at first not pronounced or severe, but seems more like lumbago than anything else. It may be paroxysmal; it is increased by motion, especially by climbing stairs or other ascents. It is frequently reflex, passing along the course of the lumbar plexus, shooting down into the scrotum or ovaries or into the thigh- or knee-joint like the pain of morbus coxæ. It is cumulative, getting worse, more constant, and more definitely located from day to day until the suffering becomes intense and constant. I am now speaking of a typical or average case. But there are more acute cases in which the pain is intense and positively localized from the beginning, and there are cases essentially chronic which never suffer intense pain or other serious local symptoms.

Tenderness on bimanual pressure in the ilio-costal space is always present, and commences shortly after the advent of pain. At first it is not well defined or accurately localized, but as the case progresses both these qualities manifest themselves. The tenderness, like the pain, is progressive, getting worse as the pus accumulates and compresses the nerves involved. For the purpose of relieving muscular pressure the patient is apt to lie on his back with his legs drawn up, and he walks with a peculiar stooping gait, stepping along slowly and carefully to avoid jolts and jars. As the case progresses he is com-

pelled to lie in bed, and the tenderness becomes so great that pressure is almost unendurable.

An irregular swelling in the ilio-costal space, projecting posteriorly and laterally, usually develops in proportion as the tenderness increases. At first it is tense and hard, without definite boundaries, but it soon becomes more circumscribed and gives evidence of "pointing" in the ilio-costal space. Deep fluctuation can now be detected, but it soon becomes more superficial, and all doubt as to the nature of the swelling disappears. Sometimes, however, the pus dissects its way down the course of the psoas muscle and points in the groin, or it may travel upward along the same muscle, beneath the ligamentum arcuatum, and find its way into the chest. When either of these events happens the abscess is not likely to point in the ilio-costal space.

Diagnosis.—Lumbago, certain organic diseases of the kidney, spinal disease, morbus coxæ, and psoas abscess may be mistaken for perirenal abscess.

Lumbago presents neither fever, tumefaction, nor tenderness that is worth mentioning; it is not accompanied by reflex symptoms; the lameness is characteristic and entirely unlike the stooping helplessness produced by perirenal abscess.

Renal lesions like pyonephrosis, cancer, tubercle, and calculus have definite and well marked symptoms which point to the kidney itself. They have already been described, and the reader is referred to the foregoing pages for details.

But it must not be forgotten that any one of these affections may antedate and cause perinephritis, and consequently that they may coexist and complicate the diagnosis somewhat; but a history of the case and a careful analysis of the symptoms, together with a study of the urine, will reveal the facts.

Disease of the dorsal spinal column in the early stage has been confounded with perirenal abscess. Gibney relates a case in which a spinal brace was applied over a perinephritic abscess under the supposition that it was a case of spinal caries. The distinction can easily be made. In spinal disease the pain is referred to the anterior median line or "pit of the stomach;" the patient walks with a stiff, inelastic gait, does not stoop forward as in perirenal abscess, and turns his whole body instead of rotating the spine if he wishes to look backward or turn halfway round; later on the power of standing upright is lost and the patient falls forward, supporting himself with his hands on his thighs or some other support; the spinous processes of the affected vertebræ are tender to pressure, to the application of heat, and to percussion, and they soon become prominent from displacement backward; and along with this symptom we shall find more or less paralysis of the sphincters and muscles of the lower extremities.

Morbus coxæ ought not to be mistaken for perinephritis, but it has been and may be again.

The symptoms of hip-joint disease are lower down than those of perinephritis; there is tenderness on percussion over the great trochanter which is referred to the hip-joint, and the same thing happens when the sole of the foot is smartly tapped, the leg being held stiff; shooting pains are felt in the knee-joint, and the patient, generally a

child, is likely to be of a strumous habit. These symptoms are wanting in perirenal abscess. If, however, the pus from a perirenal abscess finds its way down the sheath of the psoas and points in the groin, as in a case which occurred in Morris's practice, the diagnosis may be more difficult, but not impossible, for there will still be absence of the history and positive symptoms of morbus coxæ, while the induration and tenderness of the ilio-costal space will point to perirenal disease.

Psoas abscess may more easily mislead the physician. But psoas abscess is likely to be due to spinal caries, in which case the history and symptoms of pre-existing spinal disease will be present; the pain and tenderness of psoas abscess will be nearer the median line than that of perinephritis; and the history of the former does not reveal the same constitutional symptoms which precede and accompany the latter. Nearly every case of perirenal abscess is preceded by some source of infection which should direct the practitioner's attention to the real source of the trouble.

PROGNOSIS.—Perirenal abscess is a serious disease, and always involves danger, except in those rare and exceptional cases which terminate in resolution instead of proceeding to suppuration. If an early diagnosis is made, if the pus does not wander up or down the track of the psoas, if no serious pre-existing organic lesions add to the danger, and if an early and thorough evacuation of the pus is secured, recovery is likely to take place. But cases where the opposite of these conditions obtain usually end, as Morris puts it, "in prolonged suffering, hectic, and death."

As a clinical fact, most cases of perinephritic abscess *are* complicated and caused by conditions which add greatly to the danger; too many cases remain undiagnosticated until the most favorable time for efficient treatment has passed, and hence the majority terminate fatally.

TREATMENT.—When symptoms of perirenal abscess are first discovered, if suppuration has not already taken place, it is well to endeavor to induce resolution by the application of cups and leeches, followed by cold applications or, if the patient be a child or an aged person, by hot fomentations. The patient should be sent to bed and kept perfectly quiet; the diet should be milk or thin broth. A brisk saline cathartic ought to be given—half an ounce of sal Rochelle will answer admirably; and an antipyretic combined with an anodyne will act beneficially. The following is both prompt and efficient:

℞. Antifebrin, ʒj;
Codeinæ, gr. iij.
Misce, fiant capsulæ No. xij.
Sig. Take one capsule every three hours.

As soon as it becomes apparent that pus has formed the case falls to the care of the surgeon. Free incision through the loin parallel to the erector spinæ, thorough antiseptic irrigation, and perfect drainage are the paramount requisites. A free nourishing diet and active supporting treatment will henceforth be necessary, and if the patient recovers it will only be after a long and weary struggle.

RENAL PARASITES.

In this country renal parasites are very unusual. Those most likely to occur are—

(1) Echinococcus;
(2) Strongylus gigas;
(3) Pentastomum denticulatum;
(4) Distoma hæmatobium;
(5) Spiroptera hominis;
(6) Ascaris lumbricoides (accidentally found in kidneys).

(1) **Echinococcus.**—By far the most common renal parasite is echinococcus, but this is very unusual in the United States, because intimate physical association with the dog, its natural host, is not common. The echinococcus "is the immature or embryonic condition of a tape-worm that inhabits the intestines of the dog" (Ebstein), and it is only in countries where the dog lives on terms of domestic intimacy with man, as in Iceland, or where the flesh of the dog is consumed by man, as in Silesia, that this invader is common enough to be of clinical interest.

History.—The ova of the tænia echinococcus, having been received into the human stomach, pass onward into the intestinal canal, where they undergo the first stage of development and become minute cysts, known as "hydatids" or "echinococci." The individual "hydatid," "echinococcus," or "scolex" is from $\frac{1}{75}$ to $\frac{1}{225}$ of an inch in length; the head is furnished with four suckers and a proboscis, around which are sickle-shaped hooklets, "the number of which varies between 28 to 36 and 46 to 52" (Kuchenmeister).

If the echinococcus remains in the human intestine, it develops into a tape-worm, or, what is more likely, it perishes and is voided with other excreta, as the human intestine is not the most favorable soil for the growth of the tænia echinococcus; if the parasite finds its way into the liver, kidney, or any succulent tissue which affords appropriate pabulum, it undergoes development—or, more correctly, degeneration—into a series of minute cysts known as "hydatids" or collectively as "hydatid disease." The cysts vary in size from a milletseed to a goose egg, or even larger; the larger cysts are known as "mother cysts," and the smaller, which spring from the inner layer of the laminated wall of the mother cyst, are known as "daughter cysts;" another progeny may develop from the wall of the daughter cyst, and still another from the third, so that four generations are occasionally found in the original hydatid. Of course many "families" may thus develop in the same kidney, and so a community of associated families constitutes what is known as "hydatid disease." The kidney ranks next to the liver as regards liability to hydatid disease, but there are five cases of hydatids of the liver to one of the kidney.

Symptoms.—All writers agree that the symptoms of hydatid disease in the kidney, as well as elsewhere, are very unsatisfactory, and no two writers describe them alike. The disease generally develops very slowly, is practically painless, produces no change in the urine until it is well advanced, does not present a palpable tumor until months or even years after the primary invasion, and hence the early stage is gen-

erally unrecognized and unrecognizable. But there are exceptions to this rule, though they be few and far between. Occasionally the cysts develop rapidly and produce painful distention of the kidney; hæmaturia follows and a tumor is quite apparent; some of the cysts rupture into the renal pelvis; the echinococci or their hooklets appear in the urine; and the diagnosis is at once established. Now and then it occurs that, while the disease progresses in its usual dilatory manner, a few cysts burst into the pelvis, and the parasites appear in the urine before any other symptoms are manifested. If these few echinococci happen to be discovered and identified, an early and positive diagnosis can be made. But the average case progresses insidiously for months or years, until at length a tumor is felt in the lumbar region. Only about half the cases, according to Ebstein, produce a tumor. When the tumor is felt, it is rounded, very tense, generally presents ovoid nodulations which project boldly from the general surface, is not tender to pressure, or at least very slightly so, and has a peculiar elastic quality quite unlike any other tumor or swelling. It is difficult to describe this peculiarity. The tumor yields to bimanual pressure in an elastic, tremulous manner, and quickly springs back to its normal shape again, like a living thing. If this experiment be repeated a few times, the trained fingers of the physician will soon recognize the sensation I am trying to describe. I have not seen this symptom mentioned, but I regard it as almost pathognomonic. If the so-called "thrill" or "fremitus" can be detected, it is of course an exceedingly valuable diagnostic sign, but as a matter of fact it is not very often recognizable. "It will be felt most distinctly when the tumor is lightly compressed by two fingers of the left hand and a slight tap given to it with the right hand, or when the finger is allowed to rest for a time upon the pleximeter after the percussion stroke" (Ebstein). It can only be felt when the "mother vesicle" encloses a great number of cysts; a single cyst will not produce it.

In most cases some of the cysts ultimately burst and shed their contents into the renal pelvis. This is followed by symptoms resembling the passage of a small calculus through the ureter, only less intense and painful. These symptoms are succeeded by the discharge of echinococci, with fragments of cysts and hooklets, *per urethram*. Similar attacks are likely to occur from time to time as different cysts or families of cysts rupture and discharge their contents. But the presence of the cysts is pretty certain to provoke pyelitis, with pyuria and profuse hæmaturia.

In some cases the cysts discharge their contents through the cortex and capsule into the perirenal tissues, and a perirenal abscess follows. Or the hydatid tumor may grow upward, push its way through the diaphragm, and open into a bronchus, when the echinococci and their remains will be coughed up, and the expectoration may have a urinous odor. It should be borne in mind that hydatid tumors of the kidney, like other renal tumors, are generally behind the colon, so that superficial percussion will be tympanitic, while percussion after firm pressure will elicit dulness. The left kidney is rather more likely to be the subject of hydatid disease than the right—a fact which has a certain diagnostic value.

Diagnosis.—Hydatids of the kidney may be confounded with "cystic degeneration or metamorphosis of the kidney," with hydro-nephrosis, and ovarian tumors.

General cystic degeneration almost always involves both kidneys—is accompanied by hypertrophy of the heart and increased arterial tension. The tumors—for there is generally one in each renal region—are soft, reniform, do not fluctuate, have not the peculiar elastic quality which I have described or the hydatid fremitus. The urine is likely to contain crystals of cholesterin; the patient is anæmic, sallow, and emaciated—an array of symptoms quite unlike those of echinococcus of the kidney.

The differential points which distinguish hydro-nephrosis from hydatids have already been given. (See Hydro-nephrosis, page 787.)

Ovarian tumors need hardly be mistaken for renal echinococcus. Ovarian tumors grow upward; they extend to or beyond the median line; they are in close relation with the uterus; they are distinctly fluctuating; they are nearly spherical and not nodulated; and they grow more rapidly than hydatids. In cases of doubt aspiration may be employed to settle the question definitely.

Prognosis.—Hydatid disease of the kidney is rarely fatal and never gets well. Many cases progress so slowly and are productive of such slight discomfort that they are never discovered unless accidentally revealed *post-mortem* after death from some other cause. If the cysts burst and discharge their contents into the perirenal tissues, the resulting abscess may become a source of danger. If they escape into the pelvis and are discharged *per urethram*, troublesome inflammation of the urinary tract may follow, but these complications can generally be controlled by timely and appropriate treatment.

Generally speaking, the resources of surgery are competent to deal with echinococci of the kidney and the complications resulting therefrom.

Treatment.—Medical treatment is of no avail whatever, since there are no tæniacides which will kill the larvæ while they infest the alimentary canal, even if their presence were known; and the same may be said with greater emphasis after the parasites have reached the glandular organs. Aspiration, tapping, electro-puncture, and the injection of germicides, such as alcohol, iodine, and mercuric bichloride, are uncertain and to a certain extent are dangerous. The only means of cure which amounts to anything is nephrotomy, the removal of the echinococci by curetting and irrigation, and subsequent package and drainage. This is a comparatively safe procedure, and, if thoroughly done, will almost certainly result in a cure. For detailed technique the reader is referred to works on renal surgery, especially the excellent monographs of Henry Morris and W. Bruce Clarke.

(2) **Strongylus Gigas.**—This parasite is exceedingly rare in man; few American physicians have met with cases. It is more common among the lower animals, especially "the dog, the wolf, the horse, the ox, the American marten, and in some other animals. The strongylus gigas in its external appearance bears great resemblance to a large earth-worm. The female is longer than the male—in specimens taken from the dog respectively 31, 36, and 64 centimetres (12, 14, and 25

inches). It is distinguished from the ordinary ascaris lumbricoides by its reddish color, which evidently is produced by the bloody fluid in which it is usually found; further, by its great size and the presence of six nodules or papillæ around the mouth, ascaris lumbricoides having only three of these" (Ebstein). It is found in the renal pelvis, where it produces great irritation, with pain, hæmaturia, pyrexia, and strangury.

The SYMPTOMS bear some resemblance to those resulting from calculus, except that the pain is not so intense, not so sharply localized, but is more constant.

It is not known how the worm reaches the kidney. A refined differential DIAGNOSIS is impossible, but the anomalous character of the symptoms might raise a suspicion of some foreign body in the kidney, and the process of exclusion might lead to a conjectural diagnosis.

No medical TREATMENT will destroy the parasite or produce any material influence thereupon.

The symptoms may be so urgent that the surgeon will be induced to perform nephrotomy, which will result in the eviction of the worm and cure of the patient.

(3) **Pentastomum Denticulatum.**—This is a parasite belonging to the order of "mites," whose natural habitat is the nasal cavity of the dog, but which by some mysterious migration has been known to find its way to the human kidney. It is a pathological curiosity, but has no clinical interest, having been found but once or twice in man. It produces no recognizable symptoms when located in the kidney, could not be successfully treated if recognized, and may be dismissed with feelings of gratitude that it is not likely to increase in pathological interest so far as man is concerned.

(4) **Distoma Hæmatobium.**—This parasite, which was first discovered by Bilharz, and by some writers called "Bilharzia hæmatobia," is also a pathological curiosity rather than a clinical verity, so far as the temperate latitudes are concerned. It is very common in Egypt and Southern Africa. It is unisexual; the female being almost three quarters of an inch in length, while the male is not more than half an inch.

The embryos enter the alimentary canal through the medium of drinking water; thence they make their way to the kidneys, where they mature and deposit their eggs, which undergo development up to the embryo or larval stage, after which, says Ebstein, "their further destinies are unknown." They do not develop in the urine or in impure water, but in salt water or pure fresh water they are liberated, change their form, and swim freely about. They sometimes block the ureter and cause hydro-nephrotic distention above. If they enter the bladder, as they sometimes do in large numbers, considerable irritation will follow.

The distoma induces no special or characteristic SYMPTOMS, and the only certain means of DIAGNOSIS is the discharge of the ova in the urine.

Of course no TREATMENT can be adopted that will have any influence on the parasite.

(5) **Spiroptera Hominis.**—This parasite "must be mentioned in order to warn observers against confounding it with animals acci-

dentally present in the urine or placed in it designedly by malingerers, and which may be mistaken for parasites of the urinary organs" (Ebstein).

(6) **Ascaris Lumbricoides.**—It sometimes happens that the common round-worms of the intestinal canal find their way into the renal pelvis—just how it is difficult to explain. They have been mistaken for the strongylus gigas, but the invariably deep red color of the latter should be sufficient to enable the practitioner to escape this error.

ABNORMALITIES OF FORM AND POSITION OF THE KIDNEY; RENAL INADEQUACY.

By JAMES TYSON, M. D.

ABNORMALITIES IN NUMBER, SHAPE, AND POSITION OF THE KIDNEY.

Absence of the Kidney.—Total absence of both kidneys occurs only in cases of extreme defect of development, and is incompatible with continued life.

Congenital absence of one kidney is not very rare, the missing one being usually the left. It may be suspected when over the normal situation of a kidney a tympanitic note only can be elicited by percussion; yet an over-distended colon may produce such note, while commonly the necropsy first informs us of the absence of a kidney. In such event the remaining kidney supplements the work of the missing one, and serious consequences only result when the remaining organ becomes diseased or injured or is removed by operation. The ureter and pelvis of the absent kidney are wanting also, but sometimes the remaining organ has two pelves and two ureters. Occasionally the rudiment of one ureter is present. Congenital atrophy of one kidney is more common, but it cannot be recognized before death.

Lobulated Kidney.—The most usual anomaly in the shape of the kidney is the persistence of the lobulation natural to the fœtal state at the end of eight weeks of development. A minor degree of such lobulation is maintained for a short time after birth, but disappears usually during the first year of infancy. The fissures separating the lobules are not usually deep, but occasionally they are markedly so, dividing the kidney into separate segments, such as are more frequently seen in the lower animals. From seven to twenty renculi are found in the state of permanent lobulation.

Horseshoe Kidney.—The most striking of the anomalies of form is known as the horseshoe kidney, in which usually the lower ends of the two organs are united, either by true renal tissue or by a band of fibrous tissue. Much more rarely the middle or upper parts of the organs fuse, more frequently the former. In any event, this coalescence is apt to be associated with displacement of the organ. It is always placed lower down than the normal kidney, usually just above the promontory of the sacrum, less often in the pelvis, and at times on either side of the spinal column. In the fused kidney there are usually found two pelves with two to four short ureters; more rarely there is but one ureter. The ureters pass over the front of the kidney. The

arteries which go to the kidney spring from the aorta at points corresponding to the situation in which the organ is found. Thus, when above the sacrum, the arteries spring from the back of the aorta near its bifurcation or from one of the common iliac arteries, while the veins enter the corresponding parts of the vena cava or iliac veins. The horseshoe kidney is generally also found first at autopsy, but can sometimes be recognized in its abnormal position above the sacrum, especially in thin persons.

Displaced Kidney.—The kidney of normal shape is occasionally displaced. More frequently the left is so displaced, and it may lie on the lumbar vertebræ, the sacrum, or in the inguinal canal.

Movable or Floating Kidney.—The most interesting deviation of situation is found in the so-called floating kidney. The organ exhibits very different degrees of mobility in different instances. It may be so slight that it can only be recognized by expert manipulation. Sometimes, on the other hand, the kidney may be easily grasped by the hand through the abdominal walls. In the latter case there is a mesonephron or peritoneal fold loosely attaching the kidney to the spine.

Etiology.—The movable kidney is more common in women than in men, six times as frequent in the working classes. It is far more frequent on the right side than on the left. It is held by some that one fourth of all women have movable kidneys. The repeated pregnant state has been held responsible for the more striking instances of movable kidney, but I have found it as often at least in unmarried women. Mechanical violence, as by falls, has also been followed by it. It is more likely, however, that the majority of the floating kidneys are congenitally loose, while it is probable also that this looseness may be increased by the conditions named, to which should be added tight lacing. It is much more often found in thin than in fat individuals, though this may be due to its easier recognition in the former.

Symptoms.—The floating kidney often gives rise to no symptoms, while those which it causes are largely reflex in character. These include obstinate indigestion of every grade, distention of the bowels with gas, palpitation of the heart, cardialgia, neuralgic pain in almost any part of the body, but more particularly in the abdomen and cardiac region, irritable bladder, and even dysmenorrhœa. It is an interesting fact that where the degree of displacement and the mobility are more marked the reflex symptoms are commonly less so. This is not without a parallel in other diseases, and in illustration may be mentioned the well known fact that prolapse of the uterus of moderate degree is often accompanied by marked reflex symptoms, while a complete procidentia usually gives rise to local annoyance of a trifling character. The direct result of the displacement, so far as appreciable, is a sense of dragging or weight, which especially manifests itself while standing, walking, riding, or dancing. To this may be added a variable amount of pain.

More serious symptoms sometimes manifest themselves as the result of torsion of the ureter, occasioned by complete rotation of the kidney, when the renal vessels and nerves are also involved. The result is agonizing pain and scanty urination, associated with symptoms of collapse, such as nausea, an anxious expression, and cold sweats. These are caused in part by obstruction to the ureter and the backing of the

urine toward the kidney. They are, in fact, the symptoms of nephritic colic. Acute hydro-nephrosis may also be the result of such strangulation. This condition ends sometimes as suddenly as it appears. Both hemorrhage and albuminuria are reported as consequences of the same condition. The former is certainly rare. Inflammatory bands may also produce strangulation.

There may be other effects of displacement due to the location of the organ at the time, of which irritation from pressure upon the bladder may be one. It is often uncomfortable for the patient to lie on the side opposite that of the displaced organ.

DIAGNOSIS.—This is variously difficult. The kidney exhibits some motility in health, descending often half an inch with each deep inspiration. Movable kidneys are sometimes so loose and movable that they may be felt through the abdominal walls with ease. Between this ready recognition and that which requires the highest manipulative skill of the examiner there is every degree. At the present day movable kidney is regarded as a much more frequent condition than formerly. So frequently has the set of reflex nervous symptoms described been found associated with movable kidney that their presence should always suggest an examination for such an organ. The examination is variously made. More frequently the patient is placed in bed on the back, and conveniently near the edge on the right side. The examiner places the fingers of his left hand flatly below the last rib at its junction with the erector spinæ muscles. The right hand is placed on the abdomen opposite the left. Strong pressure is exerted by the latter, when, if easily recognizable, the kidney may be felt. Its recognition may be facilitated by having the patient take a deep breath—to hold, and then suddenly expire, when the kidney may be felt to slip under the fingers.

Charles P. Noble suggests the following: The patient takes the standing posture, bending slightly forward, the hands placed on a table, resting a part of the weight on it. The clothing should be thoroughly loosened. The right hand of the examiner is then placed in front, immediately next the skin below the hypochondrium, the left over the lumbar region. The patient is directed to respire deeply and regularly and to relax herself during expiration. The region between the two hands is carefully palpated, when, if there is marked degree of displacement or rather of lowered position, the organ can be felt as a firm, smooth, oval body, somewhat sensitive to pressure, which also produces a sickening pain quite characteristic. More rarely the pulsation of the renal artery can be felt. The right kidney naturally moves with the breathing more than the left, being pushed down by the liver. Sometimes the manipulation will be more successful in the knee-elbow position. When in this position, the movable kidney having fallen forward, a resonant note may be obtained by percussing over the normal situation of the organ. The displaced organ is hardly likely to be confounded with anything else. The spleen, which corresponds nearly in size, is also sometimes movable. Its shape is, however, different. Its anterior border is sharp and often notched. Sometimes both the left kidney and the spleen are floating. A movable pyloric tumor has been mistaken for a movable kidney. The passage of a stomach tube in cases of doubt would settle the diagnosis in the last instance.

TREATMENT.—As may be inferred, many cases of movable kidney require no treatment. In a few cases the symptoms are removed by improving the general health of the patient; in others the patient is comfortable while lying on the back, and such comfort may continue for a time after rising, and the rest cure may secure further relief. But when decided symptoms attributable to the floating kidney are present, surgical treatment for fixing the kidney—nephrorrhaphy—is alone of permanent value, and this sometimes fails. Nephrectomy affords a more certain relief to the annoying symptoms. I have known an operation for the removal of the kidney to be necessary after nephrorrhaphy had been attempted twice unsuccessfully. The use of pads and supports has never proved successful in my cases.

RENAL INADEQUACY.

The term renal inadequacy was applied by the late Sir Andrew Clarke to a class of cases in which the main feature is that the kidneys appear to be able to excrete no more than the normal daily quantity of urine, which yet has a low specific gravity, 1002 to 1008, and is deficient in urea, containing not more than 2 per cent., though the uric acid is natural. These patients, according to Sir Andrew Clarke, do not pass a larger quantity of urine even though they drink freely of water. The urine in such cases is, however, devoid of albumin and there are no casts.

On the other hand, the term renal inadequacy may with propriety be applied to any condition of the kidneys in which the amount of solids eliminated in the twenty-four hours is below the normal amount, regardless of the quantity of urine, although in my experience the urine is usually scanty. The specific gravity is generally high, because of such scantiness. The quantity ranges from, say, 20 to 30 ounces in the twenty-four hours, with a specific gravity constantly exceeding 1025. To this may be added the fact mentioned by Sir Andrew Clarke that it is almost impossible to get these patients to excrete more urine, even if they be given water, including the alkaline mineral waters, in considerable quantity. Such patients often also may have the symptoms of lithæmia as contrasted with gout.

TREATMENT.—The condition is best met by a diet containing a minimum of nitrogen, including potatoes, rice, and the succulent vegetables, such as tomatoes, beans, and peas, together with the free use of alkaline mineral waters. I know of no true alkaline mineral waters native to this country east of the Rocky Mountains. They have their type in the waters of Vals and Vichy in France. The numerous negative mineral waters so highly vaunted in this country are useful as diluents, but no further. To this extent they have a certain value, but I prefer to them the use of equal bulks of pure or distilled water, to which alkali in the shape of sodium bicarbonate or potassium citrate, in doses of a drachm to two drachms a day, may be added. The preparations of lithia, such as the citrate and tartrate, may also be added to such water with advantage. To such measures should be conjoined outdoor life, active exercise. Turkish baths, frictions, and a hygiene generally wholesome are good adjuvants.

DISEASES OF THE BLADDER AND PROSTATE GLAND.

DISEASES OF THE BLADDER AND PROSTATE GLAND.

DISEASES OF THE BLADDER; PROSTATITIS.

By I. N. DANFORTH, M. D.

DISEASES OF THE BLADDER.

Acute Catarrhal Cystitis.

The bladder is an intolerant organ, promptly resenting infective invasions or instrumental rudeness by the manifestation of active and unmistakable symptoms.

Etiology.—The excitants of acute cystitis mostly reach the bladder through the urethra. Perhaps the most common cause is the unclean catheter or bougie—a cause which is unnecessary, and therefore unpardonable. Gonorrhœal infection is responsible for another large group of cases, the infective germs making their way into the bladder by migration or being swept into the organ by urethral injections propelled with too much force. In females non-specific but infective muco-pus from the vagina or vulva may pass into the bladder and provoke inflammation. Foreign bodies in the bladder—of which calculus is the most frequent example—generally cause cystitis, although the passage for diagnostic purposes of sounds which are not properly disinfected may be more directly responsible than the stone itself. The rash employment of such drugs as copaiba, sandal oil, turpentine, and cantharides has repeatedly been known to produce violent cystitis. Over-distention of the bladder by postponing the act of urination unduly sometimes produces cystitis. Toxic urine, as from a suppurating kidney, lithæmia, and oxaluria, especially when they result in crystalline deposits in the bladder, are frequent and prolific sources of cystitis. Some English practitioners believe that rheumatism or gout, especially "suppressed gout," may find expression in an outbreak of cystitis; but Sir Henry Thompson characterizes "suppressed gout" as "a very present help in time of trouble" to physicians of deficient diagnostic acumen in cases of unusual obscurity.

Pathological Anatomy.—The bloodvessels of the bladder are injected, dilated, and tortuous; the epithelial layer is swollen and softened; the individual cells are swollen and distended with thick, tenacious mucus; a little later the epithelial cells are thrown off in large flakes, after which they rapidly undergo degeneration and disintegration,

which results in setting free their nuclei; the liberated nuclei now rapidly multiply, producing pus corpuscles; meanwhile another brood of pus corpuscles is derived from the leucocytes which escape from the capillaries by "diapedesis," and from their descendants, so that the inflamed mucous membrane is covered by a thick, tenacious layer of muco-pus.

The microbes present are (1) Bacterium coli communis, (2) diplococcus, (3) staphylococcus, (4) gonococcus, and (5) micrococcus ureæ; and they are mentioned in the order of their frequency, the bacterium coli being almost invariably present.

Symptoms.—Two well defined types of acute catarrhal cystitis are met with in practice: first, a mild type, like that which follows exposure to cold or is induced by lithæmia; and, secondly, a severe, violent, or "fulminant" type, such as that which follows maladroit instrumentation or forcing gonorrhœal injections with virulent gonococci into the bladder, or sometimes follows labor when asepsis has been neglected or carelessly administered, especially if the labor has been long and tedious, accompanied by a perineal laceration with suppuration and followed by slovenly treatment. It goes without saying that *all* cases of cystitis belonging to the latter class may be and should be prevented.

Mild Type.—The symptoms which distinguish the first group of cases are frequent and somewhat urgent calls to urinate, accompanied by a dull but not very severe pain behind the pubic bone; occasionally a twinge of pain shoots down into the glans penis or the glans clitoridis, but this is neither constant nor pathognomonic; more frequently a dull ache is felt in the testicles or ovaries; there may be an ill defined "backache," but it is quite as likely to be absent. The urine rapidly becomes light colored, cloudy, then turbid, and at length begins to deposit a heavy grayish white cloud of ropy mucus, which adheres to the bottom of the vessel, sometimes with such tenacity that if it is turned bottom upward the deposit will still cling thereto. If a drop of this tenacious mucus is placed under the microscope, it will be found to contain patches of bladder epithelium in a state of "cloudy swelling," and great numbers of smaller cells which are classed as leucocytes, mucus corpuscles, or pus cells according to the views of the observer and the stage of the disease. Micro-organisms of various kinds and in vast numbers are always present in catarrhal urine after it has stood a few hours.

The urine is at first acid, but after a few days becomes alkaline, and throws down a copious deposit of triple phosphates; it generally contains a little albumin, its quantity being dependent upon the amount of pus in the urine.

There are no constitutional symptoms worth mentioning; the temperature remains normal, or, as M. Guyon has pointed out, if pyrexia develops, it is all but proof positive that some other and graver lesion than cystitis exists; and the physician should diligently seek for some adequate cause if a fever temperature appears.

Severe Type.—The symptoms of the severe or "fulminant" type are very radical. The patient is seized with intense pain, usually directly behind the pubis, and radiating both upward along the ureters and

downward through the urethra; the desire to urinate is constant and intense; urination producing but partial and transitory relief, the patient has a constant and uncontrollable desire to empty the bladder; vesical tenesmus is quickly superadded; control over the sphincter is lost, and the clothing is apt to be soiled by the forcible ejection of urine in spite of determined efforts to prevent it. The rectum frequently participates in the local excitement, and rectal tenesmus with involuntary expulsion of the contents of the bowel adds to the discomfort and annoyance of the patient. All this time the pain is intense, caused partly by the exquisite tenderness of the mucous surface of the bladder, partly by the spasmodic contraction of both bladder and rectum. The patient's condition is indeed that of intense and pitiable suffering.

The urine is generally bloody, sometimes being stained only moderately, sometimes so loaded that coagula form in the bladder, so that strangury is added to the suffering already described. Thick, ropy, tenacious mucus is secreted in immense quantities by the bladder cells; it may soon become purulent, and hence the urine is loaded with great masses of muco-pus; thus the three essential symptoms of Guyon—frequent micturition, dysuria, and pyuria—are developed with great rapidity and intensity. The urine rapidly becomes alkaline, and is loaded with phosphatic crystals; a little later it becomes ammoniacal and offensive on account of fermentative changes that occur in the bladder. Albuminuria is generally present, even before true pus is formed, but it is small in quantity and of no special pathological import.

The microscope shows bladder epithelium in large flakes and single cells, leucocytes or pus cells, and triple phosphates, entangled in clear glassy mucus.

Constitutional symptoms are conspicuously absent: the temperature is normal, digestion is not seriously disturbed, and, in fact, no general symptoms accompany the intense local signs already described.

Complications.—In the majority of cases acute cystitis is not complicated by a pre-existing lesion, unless it be chronic cystitis or some form of gonorrhœal infection, as urethritis in the male and vaginitis in the female. It is far more likely to be itself a complication of some acute infectious disease, such as scarlatina or smallpox—a fact with which every experienced clinician is familiar.

Diagnosis.—There is no difficulty in the diagnosis of cystitis *per se*, frequent urination, dysuria, and pyuria being sufficiently pathognomonic. But if it has existed for any considerable time, pyelitis is apt to be present on account of the migration of an army of microbes upward through the ureters; in *all* cases of cystitis, therefore, we should raise and satisfactorily answer the question as to the condition of the kidneys, especially the pelvic portion. The development of pyrexia, perirenal pain, and tenderness, together with slight return of hæmaturia, increased quantity of pus, which is uniformly mixed with the urine and is *not* passed in tenacious masses, together with acidity of the urine, will warrant the conclusion that pyelitis has been added to cystitis, and that what was a comparatively simple case has come to be a very grave one.

Pericystitis, a very rare disease, may be mistaken for cystitis, but the presence of pyrexia with probably occasional rigors, the deep-seated,

throbbing pain occupying the entire vesical region, the vesical tenderness upon suprapubic pressure and vaginal or rectal examination, and tumefaction or evidence of exudation around the bladder, would furnish positive proof of pericystitis, while the absence of frequent urination and pyuria would be sufficient want of proof of cystitis.

Prognosis.—Theoretically, uncomplicated cases of acute catarrhal cystitis ought to be readily and promptly cured. In practice not more than 50 per cent. fall into this fortunate class. The remaining 50 per cent. dawdle along, tampering with patent nostrums, until they become practically chronic; or they are treated too heroically by inexperienced practitioners; or the physician errs in his diagnosis; or some other reason, equally puerile and equally needless, prevents the application of appropriate and efficient treatment, and a series of complications begin which means death without much delay, or, worse yet, a life of invalidism.

Treatment.—Acute cystitis is best treated by letting it severely alone. The mild form will usually get well in from one to three weeks without any therapeutical treatment if the patient remains quiet, restricts himself to liquid diet, which should be mainly milk, drinks plenty of pure water, and keeps the bowel free by an occasional dose of Seidlitz or sal Rochelle. If there is marked vesical irritation, it is well to prescribe a quarter-grain dose of codeine three or four times a day or "*pro re nata*." An infusion of linseed or marshmallow or slippery elm bark will comfort the patient and satisfy his longings for "some medicine." Infusions of buchu, uva ursi, or triticum repens are time-honored remedies which have achieved high reputation in the past, and are of little value. If there is a copious secretion of mucus, it is well to wash out the bladder with a mild antiseptic solution according to directions to be given in the treatment of Chronic Cystitis (page 826). But the chief requisites are rest, a bland liquid diet, the free use of pure water, and an occasional anodyne like codeine, morphine, or lupuline, the latter having proved very effective in mild cases of bladder irritation in my practice.

The patient must be warned against exposure and the indulgence in alcohol or tobacco during the treatment and for a considerable time after recovery is apparently complete.

In the treatment of the severe or "fulminant" form the same principles obtain, but they must be carried out with greater severity. The patient should be sent to bed and ordered to remain there. The lower bowel should be thoroughly evacuated by irrigation or "colonic flushing," and a mild saline aperient administered; moreover, the bowels should be kept a little more than "open;" the stools should be kept in a solvent condition. The diet should be limited in quantity and altogether liquid, and of course milk is the ideal food. The patient should be encouraged to drink freely of pure water or of some demulcent drink like barley water or toast water. The pain and vesical tenesmus, which, it will be remembered, are intense, should be absolutely and perfectly relieved by anodynes, whereof the most useful is codeine, because it relieves the distressing symptoms without producing any unpleasant after-effects. The dose required will not be less than half a grain, and a grain is frequently required every two to four hours until the symp-

toms are well under control, after which the dose may be reduced 50 per cent. or more. The effect is much more prompt and satisfactory if the remedy is administered per rectum; it should be dissolved in a small quantity of warm water and injected slowly and quietly, so as not to provoke the rectum to spasmodic contraction. It will be remembered that rectal tenesmus is one of the complicating symptoms of acute cystitis; for this reason the anodyne is much more useful and efficient when given per rectum. Suppositories containing codeine, morphine, or belladonna may be employed, but codeine by injection is much more prompt and efficient. Hot fomentations over the bladder are very comforting; the hot bath, and especially the hot sitz bath, will be found very useful.

It is of little or no use to give internal remedies for the purpose of "curing" the form of cystitis under consideration: they will be much more likely to irritate than to placate the angry bladder. Local applications, such as the passage of bougies or irrigations, are not to be thought of, as they will surely do more harm than good. If strangury or retention from the pressure of blood clots in the bladder or urethra occurs, it will of course be necessary to take measures for its relief: a velvet-eyed Nélaton catheter, No. 10 or 12 of the American scale, should be rendered thoroughly aseptic and lubricated with white aseptic vaseline; it should then be introduced gently—not so rapidly as to provoke the urethra to contract spasmodically, and not so slowly as to tire both urethra and patient; after the catheter has reached the bladder a little delay is advisable for the purpose of allowing the organ to become accustomed to its presence; then slowly inject eight ounces of a "normal" salt solution:

℞. Sodii chloridi,	ʒj;
Aquæ destillatæ,	℥xvij.

Allow this to escape, and repeat the process until the blood clots are broken down and evacuated. No other treatment will be required in an uncomplicated case of "fulminant" cystitis, but the means already recommended should be persisted in until the distressing symptoms are thoroughly and finally relieved.

Acute Croupous Cystitis.

Etiology.—Croupous cystitis bears the same pathological relation to catarrhal cystitis that croupous laryngitis does to catarrhal laryngitis. Croupous cystitis is very rarely seen, however, or perhaps it would be more correct to say that it is very rarely recognized. It has been known to follow the employment of cantharides as a vesicant or internally; septic fevers or smallpox will sometimes produce it; puerperal infection may be sufficiently virulent to provoke exudative inflammation of the bladder, although the clinical fact remains that catarrhal cystitis is much more likely to follow either cause.

Pathologically, croupous cystitis is an acute inflammation with exudation of the fibrin factors of the blood and their subsequent coagulation or fibrillation, so as to form a dense grayish membrane—the so-

called false membrane—which lines and adheres to the vesical mucous membrane until it is dislodged by disintegration. In a typical case of croupous inflammation there is no ulceration or destruction of the mucous membrane of the bladder, but we sometimes encounter cases of true diphtheritic cystitis where extensive patches of ulcerative necrosis may result.

In the diphtheria epidemics which were formerly so common diphtheritic cystitis was by no means a rare occurrence, but the rigid asepsis and isolation of diphtheritic cases at the present day is happily rendering such needless accidents less and less frequent.

Pathological Anatomy.—The pathological anatomy of croupous cystitis is that of catarrhal cystitis, except that the fibrinous false membrane takes the place of the tenacious mucus which covers the lining membrane of the bladder in the catarrhal form. But this distinction is an important one, since it makes the difference between catarrhal inflammation *without* exudation and croupous inflammation *with* exudation. It is quite true, however, that both types of inflammation may exist in the same case, one portion of the bladder being covered with a dense layer of mucus, while another portion is lined with a true croupous or fibrinous membrane.

Symptoms.—The symptoms of croupous cystitis are not essentially different from those of catarrhal cystitis. There is the same frequency of urination, with post-pubic pain, but instead of pyuria we at first find grayish shreds in the urine consisting of interwoven fibrillæ of fibrin. But as the case progresses pus will appear in the urine along with the fibrinous shreds, and then the three symptoms of Guyon—frequent micturition, dysuria, and pyuria—will be present. The urine will contain leucocytes, epithelia, and possibly triple phosphates in addition to the membranous fragments already mentioned. Of course there is albuminuria, but the quantity of albumin is small—not more than half a grain to the litre, as shown by Esbach's tubes. Occasionally retention of urine will result from plugging of the urethra by fragments of false membrane.

Diagnosis.—The diagnosis is very easily established, the presence of the fibrinous flakes at once demonstrating the existence of croupous cystitis. If, however, a case occurs in the midst of an epidemic of diphtheria, is attended by unusual constitutional symptoms, as fever, rapid pulse, and great depression, and especially hæmaturia, and the escape of shreds of membrane from the bladder coincides, the physician should at once conclude that he has a veritable case of diphtheritic cystitis.

Prognosis.—The prognosis of uncomplicated croupous cystitis is generally favorable, although some cases assume a grave degree of severity and involve danger. If an early and correct diagnosis is established and appropriate treatment is at once instituted, the great majority of cases recover after a few weeks. But diphtheritic cystitis is a very grave disease, and the prognosis is invariably bad; yet a few cases recover after a long course of treatment, although this statement must be qualified by saying that recovery is never absolutely perfect, as far as the bladder is concerned.

Treatment.—In croupous cystitis the treatment is not essentially

different from that of catarrhal cystitis. Rest, anodynes, liquid diet (mainly milk), together with plenty of pure water, comprise the most important means at our disposal. It is possible that the detachment and extrusion of the false membrane will be hastened by alkalies like potassic citrate in doses of ten grains every three hours. It should be given freely diluted and when the stomach is empty. The same end cannot be reached by irrigating the bladder with alkaline solutions, because, while the inflamed bladder will tolerate alkaline urine, which trickles down from the kidney, it will not tolerate an alkaline solution, however gently it may be injected. Demulcent drinks are useful, particularly decoctions of barley or of flaxseed. It would be both useless and harmful to administer balsam or other stimulating remedies, at least in the early stage of this disease, since they would aggravate rather than palliate the symptoms. After the disappearance of the acute symptoms, and after the false membrane has been gotten rid of, balsamic and allied remedies will be indicated; specific indications and directions for their use will be given under the treatment of Chronic Catarrhal Cystitis (page 827).

The bowels, and particularly the colon and rectum, should be kept unloaded by suitable aperients and enemata.

When the false membrane begins to break down and disintegrate, and the acute and distressing symptoms have passed away, it is well to commence to irrigate the bladder every day or every other day. Some cases require irrigation as often as twice daily, particularly those in which there was an extensive false membrane which furnishes a large amount of débris when its disintegration takes place. At this stage of the disease irrigation can be safely practised, and 5 per cent. boric acid or a weak alkaline solution—two grains of potassic citrate to an ounce of sterilized water—may be used with great advantage.

On theoretical grounds I should advise the injection of weak solutions of pepsin (two to four grains of scale pepsin to the ounce of sterilized water) if the urine is acid, or pancreatin if it is neutral or alkaline, with the view of hastening the disintegration and evacuation of the false membrane, although I have never had an opportunity to test its efficacy.

The treatment of diphtheritic cystitis requires the use of antiseptic irrigation as early as possible. Boric acid in 20 per cent. solution answers best, although mercuric bichloride, 1 : 10,000 or even 1 : 5000, might be tried if occasion required.

It is essential to evacuate the diphtheritic membrane as soon as possible, so as to arrest the necrotic process which is sure to take place beneath it. I should feel considerable confidence in the injection of pepsin or pancreatine solution, followed after a time by irrigation with sterilized water or normal salt solution. The irrigation should be carried out at least twice a day until the diphtheritic membrane has disappeared, but the operation must be performed with the greatest gentleness and care. After the dislodgement of the membrane the further treatment must be governed by the conditions of the bladder.

It will save useless repetition if the after-treatment, or that designed to repair the ravages made by diphtheritic inflammation, be considered along with the treatment of Chronic Cystitis (page 827).

The general or constitutional treatment of diphtheritic cystitis is essentially the same as for diphtheria elsewhere—namely, tonics, stimulants, nourishing diet, and perhaps the physician will feel warranted in trying the diphtheritic antitoxin, provided always the Klebs-Löffler bacillus has been found in the urine previously. (See Vol. I. pp. 692–695.)

Chronic Catarrhal Cystitis.

This obstinate and troublesome complaint is most frequently met with in old men, although it may occur in either sex or at any period of life. One of the severest and most obstinate cases I ever saw was in the person of a boy of eight years of age.

Etiology.—Many cases are the continuation of an acute attack. Hypertrophy of the prostate, deep-seated urethritis, stricture, and even the invasion of the bacillus coli communis, may cause chronic cystitis in men without any previous acute stage. In women utero-vaginal discharges may communicate bladder infection, and it is a well known fact that women of the town are frequently the subjects of chronic cystitis. Of course traumatic causes, like foul catheters, blows, falls, or septic operations, are equally operative in either sex. There is also a group of cases in which no satisfactory cause can be determined, although since Tuffier's discovery of the frequent presence of the colon bacillus in the urine it becomes more than probable that a proportion at least of the doubtful cases may be assigned to that cause.

Pathological Anatomy.—The whole bladder is thickened, indurated, and thrown into corrugations or folds; its capacity is very much reduced by contraction, so that sometimes it will not contain more than an ounce of urine. The mucous membrane is mottled or variegated, some portions being of a bluish or slate color, while other portions are red or purple; the bloodvessels are enlarged and tortuous, and sometimes the veins become varicose or sacculated. Patches of ulceration are seen here and there; in males the prostate is usually enlarged, and frequently to an enormous extent. The internal surface of the bladder is covered with dirty grayish muco-pus and incrustations of triple phosphates; not infrequently an unsuspected or undetached calculus will be found ensconsed behind the enlarged prostate gland or encysted in a fungous outgrowth of the hypertrophied mucous membrane. The observer will be impressed with the great thickness of the bladder, its leathery hardness and inflexibility, and its very slight storage capacity—changes which render it altogether unlike the normal bladder. But now and then a case occurs in which the bladder wall is abnormally and dangerously thin and fragile or atrophied, while the organ is greatly dilated, with loss of contractile or expulsive power. Such cases are usually not caused or preceded by an enlarged prostate, and in those which I have seen no definite cause could be given.

Symptoms.—*Frequent micturition* is always present; the calls to urinate are sometimes incessant, especially when the patient is on his feet or attempting to prosecute his business. In other cases the demands are less frequent, averaging from half an hour to two hours apart. When the patient is in bed the calls are less frequent, but are rarely more than two hours apart, so that the hours of sleep are much

disturbed. Now and then we meet with a patient who gets no respite, day or night, but whose life is made miserable by the tormenting demands of his inflamed and irritated bladder.

Dysuria is almost as constant as multiple urination—not quite. Yet it is so nearly constant that Guyon regards it as one of the pathognomonic symptoms of cystitis. It varies in degree from a slight and easily borne discomfort to an acute and distressing pain that taxes the resolution of the patient. It is variously located—sometimes behind the pubic bone, sometimes in the neck of the bladder, sometimes down the urethra and concentrating in the glans penis, and in some cases it shifts from one point to another or appears in two or more points at once. It is generally aggravated by exercise, by distention of the bladder, by intemperance in eating or drinking, and it sometimes varies greatly from day to day without any known cause or any apparent variation in the type or severity of the cystic lesion.

Pyuria—the third cardinal symptom of Guyon—is always present. The quantity of pus varies greatly in different cases, and even in the same case at different times. Generally it is considerable, and sometimes it is enormous. I have seen cases in which the urine contained pus to the amount of 25 per cent. of its volume. It is usually in the form of muco-pus or coherent masses of gelatinous, ropy matter which adheres tenaciously to the commode. But sometimes it is in small amount, but thin and offensive. The urine is generally alkaline, and either ammoniacal when voided or rapidly becomes so, and is strongly malodorous. The patient's person and clothing emit an offensive urinous odor which is of itself almost diagnostic of chronic cystitis. The urine contains albumin in proportion to the amount of pus; if more than that, it suggests at once the existence of some renal lesion as an explanation.

There is frequently suprapubic tenderness, and digital examination per rectum in the male will almost always reveal an enlarged and tender prostate, with tenderness of the bladder and deep urethra, while digital exploration per vaginam in the female will be likely to reveal some uterine or ovarian or other pelvic disease, which may explain the presence of cystitis. In both sexes hemorrhoids or rectal ulcer, or both, are likely to occur along with an old and very chronic cystitis.

There are no constitutional symptoms of any moment. Digestion is not likely to be much disturbed; the circulation is not affected, and the temperature remains normal. I have often been surprised at the equanimity with which the system would tolerate a long standing and irritating cystitis, even when accompanied by great local suffering.

Complications.—Hypertrophy of the prostate, uterine or ovarian disease, urethral stricture, vesical tumors, vesical calculus, or disease of the renal pelvis are the most common complications of chronic cystitis, and are also frequently causes thereof. In a given case the complicating lesions should be diligently sought for, as it sometimes happens that the discovery and removal of the complication pave the way for the cure of the cystitis, when it would be altogether incurable unless this end is first attained.

Diagnosis.—There is no difficulty in recognizing a case of chronic cystitis: the symptoms are decided, outspoken, and positive. The fre-

quency of urination, the pain attending the act and between times, together with the presence of pus in the urine, at once establish the diagnosis of cystitis. The real question, then, is not whether the patient has cystitis, but whether he has any associated lesions which complicate cystitis and have a direct bearing on the question of prognosis.

Referring to the complications just enumerated, we observe that an hypertrophied prostate can at once be recognized by rectal examination; but previously thereto the rectum should be thoroughly evacuated by an enema containing boric acid or some other disinfecting agent, so that a free and thorough exploration can be made not alone of the prostate, but of the rectum itself and the seminal vesicles as well. Vesical calculus should be sought for with a surgically clean sound manipulated by surgically clean fingers; and the same remark applies to urethral stricture.

Tumor of the bladder, including tubercle, can usually be discovered by bimanual touch—that is, with the right index finger in the rectum or vagina and the fingers of the other hand pressing above the pubis. The careful use of the sound will also help to decide the question, as it will impinge upon the tumor and betray its existence. There will also be present the usual symptoms of a vesical tumor, which will be recorded farther on. In many cases the cystoscope—to which I shall allude presently—will give positive information and settle the question at once. In the female, digital exploration of the bladder can be employed and the presence or absence of a tumor settled positively. A vaginal examination will of course reveal any uterine or ovarian or other pelvic disease peculiar to the female which may cause or increase an obstinate cystitis.

Pyelitis frequently exists along with, and in fact incites, cystitis, but the former is very often masked or overshadowed by the more positive and urgent symptoms of the latter. It is important, therefore, in every case of chronic cystitis that pyelitis be discovered or excluded.

The reader is referred to the foregoing article on Pyelitis for a detailed description of that disease, but it may be well to advert to the chief diagnostic points in this connection. In case of pyelitis or any of the allied suppurative lesions of the kidney there will be pain of a deep, throbbing, and quite persistent character in the affected kidney; this pain will be sharply localized, so that its location in the kidney need not be doubted; the affected kidney will be tender to pressure and it will usually be considerably enlarged; the systemic reaction will be marked, as evidenced by pyrexia, rigors, impaired digestion, coated tongue, and a facial expression clearly indicating sickness and suffering. All these symptoms are absent in cystitis unless the kidney has become infected and involved in the suppurative process which may have commenced in the bladder.

In my own experience pyelitis has been frequently found existing along with chronic cystitis, and in several instances I have found both kidneys implicated and the ureters very much dilated, tortuous, and thickened; and I add very unwillingly that I have had several such cases sent to me where "cystitis" was the only diagnosis arrived at by the attending physicians. An early and correct diagnosis would have been followed by nephrotomy, and probable recovery of the patient.

PROGNOSIS.—An uncomplicated case of chronic cystitis may recover; a small proportion of such cases do recover; but every physician of experience in diseases of the urinary organs knows that perfect recovery is the rare exception. Nevertheless, better results have been reached of late years because of the improved methods of treatment; and it is, I believe, certain that still better results await us in the near future. Cases which are complicated by prostatic hypertrophy, pelvic lesions in the female, or suppurative disease of the kidney are practically incurable unless the complication can be abated, and this is frequently impracticable. So also cases complicated by tuberculosis of the kidney or bladder or by tumor of the bladder must be relegated to the group of incurables. Nevertheless, much may be done to lessen suffering, procure comfort, and prolong life in the most hopeless cases. In fact, in no other disease can the physician accomplish more in the way of comforting his patient and rendering life not only endurable, but desirable. And this is particularly true of old men who suffer the torture arising from hypertrophic prostatitis, and whose lives were, not many years since, but a long-drawn period of misery, but who can now be relieved of much of their sufferings by a judicious and persistent application of modern methods of cystic sanitation.

TREATMENT.—In a simple or uncomplicated case of chronic cystitis the treatment consists of rest, an appropriate diet, the free use of pure water, irrigation of the bladder, and the long continued administration of certain so-called—and very appropriately called—"alterative remedies."

Bodily rest is important, and the patient should, if possible, spend a part of his time in the recumbent position. But this must not be allowed to degenerate into a habit of laziness. On the contrary, he should take some gentle out-door exercise every day in pleasant weather, and walking is the most beneficial form of exercise. He should be warmly dressed, and he should particularly guard against damp or cold feet and legs. My patients with chronic cystitis are advised to wear wool or part-wool underwear the year round, varying its thickness and weight according to the season. Thick shoes and stockings are also insisted upon.

The diet should be mainly milk, but an exclusive milk diet cannot be insisted upon or recommended. Patients with chronic cystitis are generally people past middle age; they are usually anæmic and have feeble or impaired digestion; hence a mixed, nutritious, constructive, and easily digestible diet is a paramount necessity. Milk may be taken as freely as it can be digested, but if it be forced upon the unwilling stomach a mass of heavy curds will be the result, and the patient will then turn from milk with well founded disgust. Yet if its importance as an article of food be laid before him, he will presently learn how, and when, and how much his stomach will tolerate, and I have had many patients who learned to depend upon milk as their dietetic mainstay, but who at the beginning were unwilling to consider it at all. A full meal of meat and vegetables should be permitted once a day, and the patient should be allowed his choice of beef, mutton, fish, poultry, or game. In fact, these patients *must* be well fed and nourished, both on account of their debility from senile anæ-

mia and the wear and tear incident to a vexatious and harassing cystitis.

In younger and more vigorous patients a more restricted diet may be advisable, and of course can be safely adopted. The plentiful use of pure water is important. The water must not be only barely and tolerably potable; it must be pure. Boiled and filtered rain water does nicely; distilled water is excellent; some of the spring waters in the market are useful, as the Waukesha and Poland springs. The so called "medicinal" waters, with their formidable list of cure-all drugs, certified by analysts of more or less repute, should be avoided. What the patient needs most is pure water which contains no medicine, and of which he should drink three or four pints daily. Patients who drink well or spring water containing lime in any of its combinations should have the water boiled and filtered before drinking it; otherwise it will be very likely to prove an irritant to the bladder, and it will be likely to lay the foundation of renal or cystic calculus.

Irrigation of the bladder must be employed systematically and persistently. After many trials of many methods I have become persuaded that a soft catheter of No. 10 or 11 calibre (American scale), and the Van Buren pear-shaped, flexible rubber syringe holding eight fluidounces, make the best apparatus for bladder irrigation. This apparatus is simple, inexpensive, easily kept clean, and the patient can be taught to use it himself, which is a matter of great importance, especially in country practice. Of course the physician must not forget that the apparatus must be strictly antiseptic when used, and that the syringe must be absolutely full of the irrigating fluid; that is, all the air must be displaced by the fluid, else air holding germs in suspension will be injected into the bladder and the patient will be harmed rather than helped.

The question will at once arise, What shall be used as an irrigating agent? In an uncomplicated case of chronic cystitis sterilized water holding in solution chemically pure sodium chloride (aquæ ℥xvj, sodii chloridi ʒj) answers admirably. Another excellent remedy is boric acid ʒij, aquæ ℥xvj. In fact, almost any mild antiseptic solution will be found useful. Sir Henry Thompson's soothing mixture answers admirably; it is—

℞. Boracis, ℥j;
Acidi borici, ℥j;
Glycerini,
Aquæ, *āā*. ℥ij.—M.

Sig. One half ounce to be added to four ounces of warm water for each irrigation.

Listerine (ʒiv, aquæ ℥xvj) and borolyptol in the same proportion are very efficient. If the bladder is very irritable, I sometimes use a solution of codeine (codeinæ, gr. j, aquæ, ℥j), and find it not only soothing and comforting, but curative. Fenwick recommends iodoform emulsion:

℞. Iodoformi, gr. x;
Mucilaginis acaciæ, freshly made, q. s.;
Aquæ, ad ℥j.—M.

Sig. Two tablespoonfuls to be injected into the bladder and left in, once daily, after the bladder has been thoroughly washed out with dilute Condy's fluid.

This author adds in a foot-note,[1] "Stop iodoform directly the patient tastes it in his saliva." My experience with iodoform emulsion has seemed to demonstrate the superiority of codeine.

When the urine is intensely alkaline and offensively ammoniacal nitric acid should be employed, ten minims to the pint. It will cause some pain, and it should be followed by irrigation with pure warm water. In some patients not more than five drops to the pint should be used. Phosphoric and acetic acids are also recommended by Morris. Nitrate-of-silver injections (one grain to four ounces of water) will be very useful in indolent cystitis attended by profuse secretion of muco-pus. The bladder should be washed with pure water for the purpose of dislodging the layer of mucus which covers its surface before using the silver. Mercuric bichloride (1 : 10,000) is sometimes employed with good results in cystitis with offensive urine. It sometimes causes severe pain, however, which of course will preclude its use. I have had much better success by using nitrate of silver once or twice a week with boric acid, or Sir Henry Thompson's mixture on other days. In many cases acetate of lead (plumbi acetatis gr. j, aquæ ℥iv) does much good; in other cases tannic acid (acidi tannici glyceritis ℥j, aquæ ℥iv) works admirably.

Many other remedies have been recommended from time to time, but the above list is already long enough. In a given case experience coupled with careful observation must determine what remedy answers best. Moreover, it will be necessary to change occasionally, since, as Fenwick observes, "the bladder often becomes apathetically indifferent to any form of wash which is continued long." Not more than four ounces should be injected at once, and at first not more than two ounces will be tolerated. At each sitting the organ may be injected twice; that is, four ounces may be injected and allowed to escape after from one to five minutes—and then the operation may be repeated. Everything should be done tenderly and gently, because an inflamed bladder is a very sensitive organ and unnecessary haste and rudeness may inflict great suffering.

There are various remedies which act the part of "alteratives" to the inflamed bladder, and which are of great value. The most valuable, in my estimation, are oil of sandalwood, copaiba, Canada balsam, and Peruvian balsam, mentioned in what I regard as the order of their value. They may be given in capsules containing five minims each four or five times in the twenty-four hours, and I always direct that they be taken on an empty stomach—namely, on first rising in the morning, an hour before luncheon, an hour before dinner, and at bed-time, and if the patient happens to awaken in the night he may take a

[1] *Cardinal Symptoms of Urinary Diseases*, p. 85.

fifth capsule then. I am now using with very satisfactory results capsules containing five minims of oil of sandalwood and one minim of guaiacol; and the latter remedy may also be combined with either of the other balsams. Canada balsam has not received the credit to which it is justly entitled in the treatment of urinary diseases, and especially cystic inflammation. I have found it particularly valuable in old, indolent, torpid cases, where a quietly stimulating influence seemed needed. Some recent trials of Peruvian balsam in cases of suppurative inflammation of the bladder with stinking ammoniacal urine have increased my confidence in this old and almost obsolete drug.

Another group of remedies which have an alterative influence, although in a different way, are the antiseptics, as represented by boric acid, resorcin, salol, thymol, and especially guaiacol. The first three mentioned may be given in doses of five grains four or five times a day, always on an empty stomach; they should always be given in gelatin capsules, recently filled, lest the medicine become hardened into an insoluble mass. The dose of thymol should not be more than two or three grains. Guaiacol cannot be given in doses of more than two or three minims, and many patients complain of nausea and flatulence if more than one minim is given. Yet it is a urinary antiseptic of great value, and should not be neglected. The carbonate of guaiacol may be employed in five-grain doses. In a given case whichever remedy is employed should be persistently given, even for several months, if it agrees with the patient and seems to be doing good. If the doses I have advised seem small—as of course they are—let the reader remember that every dose has to pass through the kidneys in order to reach the bladder, and that large doses—especially of the balsams—are apt to irritate or even inflame the kidneys. I have seen many cases of nephritis and many cases of pyelitis which were directly traceable to over-doses of the balsams given in the treatment of the lower urinary tract.

Yet another class of remedies which have more reputation than value are triticum repens, stigmata maidis, buchu, and uva ursi. They may be given in infusion, or "tea," in the proportion of an ounce of the drug to a pint of water, and the patient should take an entire pint during the twenty-four hours. When the urine is very alkaline and offensive it is well to give ten or fifteen grains of benzoic acid four times a day, or fifteen drops of dilute hydrochloric acid or of dilute phosphoric acid in water, or infusion of triticum repens four or five times a day, suspending other remedies meanwhile.

Anæmic patients will require iron, malt, or cod-liver oil; neurotic patients may need anodynes; dyspeptic people may need pepsin; but the alert practitioner will need no hints as to these simple and everyday points, which are common to all varieties of disease and all conditions of life.

Vesical Calculus.

Although vesical calculus does not legitimately come under the care of the physician, it is nevertheless necessary that he should be acquainted with its causes and symptoms, that he may endeavor to prevent

its formation on the one hand or to recognize its presence and relieve the distress it occasions after it has formed on the other.

Etiology.—The determining causes or pathology of urinary calculi, as well as their varieties, have already been described under the head of Renal Calculi, but there are some causes that especially lead to the formation of vesical calculi. The great majority of vesical calculi begin their career as renal calculi of the "uric acid" variety. These little calculi pass into the bladder, where they form the "nuclei" of larger uric acid calculi; after a time they produce irritation of the bladder, and a sound or bougie is passed for diagnostic purposes, by means of which bacterial infection of the bladder takes place; then follows cystitis, the formation of pus, decomposition of the urine, and the precipitation of ammonio-magnesian phosphates around the uric acid calculus. Appropriate treatment may remedy the cystitis, and then decomposition of the urine ceases, and another layer of uric acid or urates is formed around the calculus; but sooner or later another attack of cystitis comes on, followed by the deposit of another layer of triple phosphates around the calculus. These events may occur again and again, and thus we have the "stratified" calculus, composed of alternating layers of uric acid or urates and phosphates. The oxalate of lime calculus has much the same history as regards primary origin: it begins by the precipitation of crystals of calcic oxalate in the renal tubules, their aggregation into a minute calculus, its transit to the bladder, where it serves as the nucleus for a stratified stone—the so-called "mulberry calculus." It is a cruel type of stone, causing great irritation and pain by its roughness and hardness, so that its projecting spicules lacerate the mucous membrane of the bladder, thus producing hemorrhage into the bladder, and often adding strangury to the other painful symptoms.

Purely phosphatic calculi are hardly ever found, but a stone may consist very largely of triple phosphates around a nucleus of uric acid or ammonic oxalate, especially in old men with enlarged prostate, followed by "residual" urine behind the prostate, which becomes decomposed, with the production of triple phosphates, which are deposited around some nucleus lying in wait, generally a minute calculus which has descended from the kidney.

Cystine calculi are very rare; Sir Henry Thompson has operated upon but 1 case; Tyson reports 2 cases in the ninth volume of the *Transactions of the Association of American Physicians;* and I have had 1 case in my own practice. Cystine calculi are always small, but their roughness and waxy consistence would make admirable "nuclei" for the growth of other varieties.

The general rule may be laid down that vesical calculi are formed by slow growth around a nucleus of some kind which had its origin outside the bladder, and that in a great majority of cases the nucleus is a minute renal calculus which has migrated from the kidney to the bladder.

Symptoms.—Pain, frequent micturition, and hæmaturia are the three most constant symptoms of stone in the bladder. Other symptoms are found in most cases; now and then an anomalous case occurs in which there are no symptoms which indicate the presence of stone, and it is only revealed by sounding or on the post-mortem table; but such cases

are so rare that they count only as curiosities. The pain varies greatly in intensity and variety. It depends very much upon the size and kind of calculus present. In case of a large smooth stone there will be a constant dull pain or ache behind the symphysis pubis and a sense of weight and dragging in the perineum, with frequent attacks of sharp, darting pain upon running, walking briskly, jumping, or any active exercise. The pain shoots upward to the kidney and arouses suspicion of renal calculus, which may in fact be present; it is sometimes felt in the testicles or glans penis or rectum, or, indeed, in all these localities; it is very marked at the end of micturition, when the bladder contracts upon the stone, but there is hardly ever a time when the patient is not conscious of pain in the bladder or adjacent parts after the stone has reached any considerable size.

If the stone be of the so-called mulberry variety, the pain is more constant and more intense; it is also increased in a much greater degree by exercise.

Frequency of urination is a pretty constant symptom, and sometimes precedes pain. Frequently patients will remember, if they are asked, that for some time previous to the beginning of pain or hæmaturia they began to urinate oftener than natural and that the calls were urgent and imperative. The frequency is increased by exercise, and in pretty direct ratio to the violence of the exercise; and it is markedly decreased by rest and the recumbent position.

Hæmaturia may possibly be the first symptom to attract the patient's attention and cause him to seek medical advice. It usually comes on after some unusual exercise, as a long, fatiguing walk, a sudden jump, a fall, or running at the top of his speed. After this he experiences a sudden and urgent desire to urinate, and is surprised to find that he is passing bright fresh blood, it may be in considerable quantity, although a free hemorrhage would at once cause a suspicion of tumor of the bladder.

The blood disappears in a day or two, the urine clears up, but sooner or later another similar attack comes on and pain and frequent desire to urinate also make themselves manifest.

Another symptom of considerable importance is the sudden arrest of the stream during urination, accompanied by tenesmic contraction of the bladder. It is occasioned by the stone being swept against or into the vesico-urethral orifice and occluding more or less perfectly the entrance to the urethra.

The urine will generally contain albumin, muco-pus, bladder epithelium in great quantities, blood globules, and urinary crystals in great number.

Diagnosis.—The diagnosis ought to give little difficulty. Cystitis, the most perplexing complication, will always be present; but the vesical sound will reveal the presence of calculus, and the physician will at once understand that the cystitis is of secondary consequence.

Tumors of the bladder are announced by free hemorrhage, appearing without apparent cause, perhaps without any pain, but certainly without the acute pungent pain of stone of the bladder.

Renal calculus will sometimes produce the bladder symptoms of vesical calculus, but the symptoms of renal calculus already described

will antedate those of vesical calculus, and if the physician will follow the history of the case back to its beginning, he will have no difficulty in arriving at a correct conclusion. It must not be forgotten, however, that the patient may have both renal and cystic calculus at the same time.

TREATMENT.—So far as the physician is concerned, the treatment is prophylactic, rather than curative. It is now quite possible for a physician to foresee the danger of calculous disorders and prevent them in people to whom he stands in the relation of medical adviser. A stone is not formed suddenly. It is invariably preceded by a series of symptoms and events denoting a "diathesis" or condition of system which not only renders the patient peculiarly liable to a calculus, but indicates what kind of a calculus he is likely to have; and, knowing this, the physician can and should institute treatment for the purpose of preventing the threatened mischief.

These "diatheses" and the treatment required in each have been described in the article on Renal Calculus (page 782), to which the reader is referred; and, as renal calculus almost invariably precedes vesical calculus, and a renal calculus is invariably preceded by symptoms indicating *its* probable formation, the reader will not fail to note that the ultimate object of treatment should be to prevent the formation of both renal and vesical concretions.

The treatment of the cystitis which accompanies vesical calculus, as well as the pain and irritation consequent thereupon, will be essentially the same as that already laid down in the article on Chronic Cystitis (page 825). It will consist of bladder-irrigation and the use of antiseptics and anodynes until the stone is removed by the surgeon—an operation which has been so much improved in recent years that few cases are beyond its benefits.

TUMORS OF THE BLADDER.

Our clinical knowledge of tumors of the bladder is of quite recent date. Up to fifteen or twenty years ago they were mainly curiosities of the post-mortem table, and interesting to the pathologist and histologist rather than the clinician. But recent methods both of diagnosis and treatment have brought them within practical reach of the physician and surgeon. It is therefore necessary that they be briefly considered in a systematic treatise on general medicine.

Mr. W. Roger Williams of London, formerly Registrar of the Middlesex Hospital, as the result of a very extensive investigation found that 0.65 per cent. of all tumors originated in the bladder.

Bladder tumors are usually located in the region of the trigone vesicæ, adjacent to the ureteral orifices. In rare instances they may grow from the lateral walls of the bladder. The form of the tumor is usually rounded or pear-shaped, with the exception of papillomata, which are flattened or saucer-shaped. Their size varies greatly: some are no larger than a chestnut; the great majority range from the size of an English walnut to that of a hen's egg. In rare cases they are multiple, two or more being found in the same bladder, but they are always malignant, and are due to infection by "contact;" the surface of the

primary tumor coming in contact with the opposite bladder wall and infecting it by contact, just as happens in other parts of the body.

Tumors of the bladder may be "infiltration tumors"—that is, so interwoven with some one or indeed all the thicknesses of the bladder wall that they cannot be separated from it; or they may be "sessile tumors"—that is, sitting upon the mucous layer of the bladder, but having no intimate connection with it; or they may be "pedunculated tumors"—that is, attached to the bladder by a stem or pedicle only.

Tumors of the bladder may be either malignant or non-malignant, although the researches of Mr. W. Roger Williams showed that the former were far more common. Out of 90 cases of bladder tumors, 59 were cancer, 6 sarcomata, 2 fibromata, 23 papillomata or villous growths. This shows that nearly three quarters of the cases of cystic neoplasms are malignant in their nature.

BENIGN TUMORS.

(*a*) *Papillomata.*—These are sometimes erroneously described as "villous cancer," but, as Ziegler remarks, "the growth is not malignant, and should not be described as a cancer."[1] As Morris points out, there are "two kinds of papillomata of the bladder—namely, the fimbriated or villous polypi, and the fibro-papillomata or the papillary tumors."[2] The former consist of a soft spongy mass made up of branching vessels, often largely dilated and tortuous, thin-walled, and covered by a layer of epithelium. These villous tumors may be single or multiple, or they may spread out like a thick bed of moss over the neck and lateral walls of the bladder. Sometimes long vascular tufts grow from these mosslike tumors which float in the urine, and are carried into the urethra far enough to cause partial occlusion; or they are cut off by the contracting sphincter, and this is followed by pain, hæmaturia, and the appearance of stringy shreds in the urine.

The papillary tumors have a basis of fibrous (or possibly muscular) tissue, which is compact, dense, and elastic, and which is surmounted by a papillary growth of looped vessels covered by a dense epithelial layer. These tumors are almost always solitary, but this rule is not absolute. They are rarely larger than a pigeon's egg, and sometimes not larger than a bean. They may be either pedunculated or sessile, but the latter condition is the most frequent.

(*b*) *Myomata*—muscular tumors—are rare, and are generally regarded as proceeding from the prostate, but W. T. Belfield has reported 2 cases which undoubtedly originated from the muscular wall of the bladder itself. They are nodular masses which very rarely perforate the mucous membrane of the bladder; hence they may exist for years without producing any marked inconvenience.

(*c*) *Fibromata* are also very rare. They spring from the submucous connective tela; are generally pedunculated; scarcely ever attain a size larger than an English walnut, provided they retain their fibroid type; they occur only in adults.

(*d*) *Myxomata* have been found in the bladders of young children, and are regarded by Morris as "in some instances a fibro-papilloma or

[1] *Text-book of Path. Anat.*, p. 784. [2] *Diseases of the Genito-urinary Organs*, p. 433.

a fibroma, the cell portions of which have undergone mucoid degeneration."

Mucous polypi have also been found in the bladder of both children and adults. I have removed one from the bladder of a man over fifty years of age, but they are so rare as to be regarded as pathological novelties.

(*c*) We also find, in rare and exceptional cases, dermoid tumors, adenomata, angiomata, and serous cysts; Bilharzia hæmatobia sometimes causes luxuriant fringed excrescences in the bladder, which bleed freely, although such cases could hardly ever occur in the United States. Along the Ganges and the Nile, however, hæmaturia is not infrequently due to this cause.

MALIGNANT TUMORS.

(*a*) *Sarcoma.*—In the *Transactions of the Pathological Society of London* for 1888, Mr Hurry Fenwick reports 50 cases of sarcoma of the bladder, with the following results: Sarcomata occur nearly always in children under five years of age or in men above fifty-five; in children they are multiple, and may be either pedunculated or sessile; in adults they are solitary and generally sessile; the average size, whether in children or adults, is that of a hen's egg, but of course in exceptional cases they may vary in either direction; they may be either round celled or spindle celled or "mixed sarcomata"—that is, a combination of spindle and round celled. Sarcoma of the bladder frequently has a villous covering sprouting from its surface, which might easily lead the inexperienced practitioner to regard it as a fibro-papilloma.

As "sarcoma originates invariably in a structure belonging to the connective tissue group" (Ziegler), it must in the bladder have its origin in the submucous connective tissue.

(*b*) *Carcinoma*—cancer of the bladder—may be either epithelioma or scirrhus, with the probabilities in favor of the former. In the majority of cases it is secondary, and, while it may follow cancer of any other part or organ, it is more likely to follow cancer of the uterus or rectum or some other adjacent part. Yet primary cancer of the bladder is more common than it was formerly supposed to be.

Cancers of the bladder assume various forms: they may form prominent tumors, pushing their way into the bladder, or large diffuse infiltrations, involving the whole thickness of the bladder and a considerable portion of its area, or deep, ragged, bleeding, rodent ulcers with elevated and indurated margins. They sometimes present villous surfaces, the villi being composed of dilated and thin-walled bloodvessels springing from the overlying mucosa. They are usually rather slow in developing until ulceration occurs, after which their progress is rapid and destructive.

All tumors of the bladder are likely to be complicated by stone, most frequently phosphatic in character, owing to cystitis, caused either by the tumor or the treatment thereof; and hydro-nephrosis or pyonephrosis is a common consequence of bladder tumors, owing to the partial but progressive occlusion of the ureters.

SYMPTOMS.—The earliest, most prominent, and most constant symptom of a tumor of the bladder is hæmaturia. It is frequently profuse—

it may be dangerously so. It is generally persistent after it begins, lasting for several successive days, or even for weeks, without cessation, although the quantity of blood passed will vary from day to day. The first appearance is usually a great surprise to the patient: it comes on without apparent cause, without any injury or extra exertion or instrumental examination, or anything else which satisfactorily explains it. It may not be accompanied by pain; in fact, it is the general rule that pain does not appear until some time after hæmaturia begins.

After the first attack of bleeding other attacks are sure to follow; indeed, the patient is liable to profuse hæmaturia at any time and without apparent cause. Yet there may be intervals of days and even weeks during which no blood appears, but these intervals grow shorter and less frequent, until no day passes without more or less loss of blood during the act of micturition. The blood is chiefly voided toward or at the close of micturition, and blood passed in this manner may always be presumed to come from the bladder. As a result of this recurrent and exhaustive hæmaturia the patient becomes pale, anæmic, and feeble, and sometimes œdema of the feet and legs from simple transudation of serum through the thin and weakened vessels occurs.

Pain is very variable as regards the time of its appearance and its severity. In nearly every case of tumor of the bladder more or less pain is felt, but it is apt to be delayed until the morbid growth becomes a mechanical obstruction to urination. Sometimes it is very intense; it reaches its acme at the end of urination, when the bladder wall contracts upon the morbid mass. It is most marked in the neck of the bladder and glans penis; it may be felt in the testicles and behind the os pubis. In cases of villous papillomata the villi are sometimes caught and pinched by the sphincter, and this causes sharp burning pain of brief duration. The character of the pain is of some value as an indication of the kind of tumor present, as I shall point out directly.

Frequent urination appears as soon as the tumor acquires any considerable size, and the frequency increases as the tumor enlarges. If the tumor in any degree obstructs the vesical orifice of the urethra, or if it encroaches upon or infiltrates the trigone, strangury will be added to frequent micturition and great suffering will result. Manual examination of the abdomen, combined with digital examination per rectum or per vagina, will often enable one to detect a morbid growth in the bladder not larger than a walnut. The rectum should first be unloaded by a boric acid enema, or, if the patient be a female, the vagina should be douched with some antiseptic solution and anæsthesia induced by chloroform or ether. The walls of the bladder can then be closely approximated by the bimanual method and a small tumor, or even a calculus, can be plainly felt. Indeed, the character, size, shape, and density of the tumor can in many cases be made out, especially in patients with thin abdominal walls. Having employed this method for more than fifteen years on many patients and under a variety of conditions, I can confirm the statement made by Mr. Henry Morris in the *Lancet* of October 31, 1896, in which he strongly recommends bimanual examination as a means of diagnosis of hæmaturia of vesical from that of renal origin, only I think this method has a wider range of usefulness than Mr. Morris gives it.

The sound is useful as a means of diagnosis of vesical tumors in very skilful and very gentle hands, but I doubt whether physicians whose surgical experience is limited, and necessarily somewhat restricted, should attempt sounding the bladder when other symptoms point to a tumor. But, if used at all, it must be only with the greatest gentleness and delicacy, and the instrument must be thoroughly aseptic. If the sound comes against a protuberance which does not yield the "click" of a stone, it will indicate a tumor; yet better evidence can be obtained by bimanual examination, with no danger of hemorrhage or septic infection of the bladder as a consequence.

The electric cystoscope, devised by David Newman and improved by others, may be employed. It requires considerable experience to acquire facility in using it, and if the bladder is filled with blood or turbid urine it will be of no use. The bladder must first be irrigated, and then partly filled with a clear solution of boric acid; the cystoscope may then be introduced, although in case of a tumor it is more than probable that the introduction of the instrument will provoke bleeding sufficiently to obscure the field of vision. Solid tumors which do not bleed, like fibromata, can be detected with great ease by means of the cystoscope when it is employed by skilful hands.

In case of female patients the bladder can be explored with the finger through the dilated urethra, and an absolute diagnosis made. The urethra should be dilated slowly and the patient should be anæsthetized.

Can we determine by any mode of examination except that of digital exploration of the bladder what kind of neoplasm the organ contains? Most certainly not; and hence I generally advise and practise suprapubic cystotomy, which can be done so speedily and safely, inasmuch as it also affords the most available means of applying efficient treatment. Yet certain deductions can be drawn from the nature of the symptoms which are of considerable value as regards differential diagnosis. A tumor which upon bimanual exploration feels soft, spongy, or flabby, like a bed of moss, which is not attended by severe pain, is not very tender upon pressure, bleeds easily and freely, and from which every now and then escape long slender shreds of tissue, is almost certainly a papilloma. At the same time it must be remembered that an epithelioma which has an outgrowth of villi may manifest some of these same symptoms, but there will be great pain, well marked tenderness on pressure, and the characteristic "cachexia" or progressive invalidism characteristic of cancer.

A tumor which is hard, round or pear-shaped, not tender, does not bleed easily or very freely, is inelastic and unyielding, grows very slowly, does not throw off any shreds or masses of decayed tissue, and is not attended by a "cachexia," is almost certainly a fibroma; but scirrhus in its early stages also produces very similar symptoms *plus* the peculiar pain, tenderness on pressure, and evidence of infection or "cachexia" peculiar to carcinoma.

A sarcoma is a bleeding tumor almost from the first; hence anæmia is an early concomitant; it is not very painful or very tender, but it has a peculiar elastic or "springy" feeling, which is almost pathognomonic to the educated touch.

By carefully sifting and weighing the symptoms, and comparing them with the life-history or career of each group of neoplasms, it is quite possible to arrive at a safe and reliable diagnosis in most cases; yet it must be admitted that now and then a case of hæmaturia confronts us in which the source of the blood cannot be determined with certainty except by the aid of the cystoscope or of cystotomy, or possibly of both combined.

Prognosis.—No case of tumor of the bladder is without danger. Malignant tumors of the bladder are nearly always fatal; sarcoma, if discovered early, may be removed, but is in great danger of recurring; small pedunculated fibromata can be safely removed; yet the clinical fact remains that most cases of vesical tumors sooner or later terminate fatally.

Since the revival and improvement of suprapubic cystotomy the number of cures has materially increased, and we may reasonably hope that greater triumphs still will be achieved in this field of surgery.

Treatment.—So far as the physician is concerned, the treatment is only palliative. Hemorrhage must be controlled by astringents, such as gallic acid, acetate of lead, or, what I esteem very highly, eucalyptus rostrata, of which half a drachm of the fluid extract may be given four or five times a day; the hæmostatic properties of ergot may be tried; pain must be relieved by suppositories containing morphine or codeine and belladonna, or if severe paroxysms of pain occur the hypodermic use of morphine may be required. Strangury may require the use of the catheter; if so, a velvet-eyed Nélaton or Jacques catheter should be employed in the gentlest manner. Irrigation of the bladder will do no real good, but if retention and strangury are caused by muco-pus or blood, it may become necessary to irrigate as a means of temporary relief. If the patient is kept awake by pain, a full dose of chloral, sulphonal, morphine, or some efficient hypnotic should be given without hesitation.

The question of relief or cure by surgical means must be relegated to the surgeon. My own experience in operating for tumors of the bladder has not been encouraging as regards resulting *cures;* but great relief from suffering and a season of comparative comfort can be secured, after other means have failed, by opening the bladder from above, so as to allow of irrigation and free drainage. Besides, if the tumor happens to be benign and pedunculated, it may be removed and a permanent cure may result.

Vesical Tuberculosis.

Etiology.—Tuberculosis of the bladder in the majority of cases is secondary to and is caused by tuberculosis of the kidney, the tubercle bacilli being carried into the bladder through the ureters. For the same reason it is frequently associated with tubercle of the ureters. It may, however, be primary in the bladder, the infection taking place through the urethra. The bacilli are many times without any doubt conveyed to the bladder by unclean catheters or bougies. Venereal infection may act as an exciting cause, or the tubercle bacilli may be received through the agency of sexual contact. Age and sex seem to

have some influence. It is far more common in men than in women, and occurs generally during middle life, when the sexual function is most active; yet it may attack children and the very aged. Sooner or later the disease is likely to invade the prostate or testicles, or both, and if it is primary in the bladder, it is pretty certain to invade the ureters and kidneys, presumably owing to the migration of the infective germs.

Pathological Anatomy.—The bladder is small and thickened, the thickening being most apparent around the ureteral orifices; diffuse masses of miliary tubercle are scattered about, being most abundant where the thickening is greatest. The mucous membrane is red, its vessels large and tortuous, and many points of ulceration are seen, some minute and superficial, others large, deep, and ragged; sometimes the mucous layer is very greatly thickened, indurated, and corrugated. The ureters are commonly enlarged, dilated, thickened, and tortuous, with here and there a tubercle colony adhering to or incorporated in their walls. The bladder is sometimes surrounded more or less perfectly with a layer of thickened, indurated fibrous tissue which renders urination very difficult and may necessitate the habitual use of the catheter.

Symptoms.—The first symptom is irritability of the bladder, as evinced by frequent desire to urinate. At first it does not attract the patient's serious attention, but soon it becomes an annoyance and he seeks advice with regard to it. A little later blood appears in the urine, the quantity being just enough to tint the urine a light pink color. But the blood disappears in a day or two, to appear again after some unusual exertion, perhaps in larger quantity, although the quantity is never very large.

As the case progresses cystitis develops and the urine contains muco-pus; along with the cystitis or shortly after pain becomes a more or less constant and harassing symptom. The severity of the pain is said to vary greatly in different cases, in some becoming mild in character and quite endurable, in others being "frequent and intense, continuous and agonizing" (H. Morris). In my own cases pain has never been a very marked symptom.

The urine remains clear and apparently normal until the first development of hæmaturia, but sooner or later cystitis sets in, and then it is loaded with muco-pus, stained now and then with blood.

The microscope will show degenerated epithelial cells from the kidney and bladder, blood and pus corpuscles, multitudes of microbes of various kinds, generally including the coli bacillus, but the true tubercle bacillus is rarely found in offensive, purulent urine. If, however, the bladder be irrigated daily for a week or ten days with a boric acid solution, so as to keep it comparatively free from pus, and *then* the sediment obtained from fresh urine by the centrifuge be examined, the tubercle bacillus will be found. In pus-laden urine it is probable that the bacilli are destroyed by the phagocytic action of the leucocytes present.

Diagnosis.—Non-tubercular cystitis requires to be differentiated from the cystitis accompanying tubercular disease of the bladder. The former generally begins with pronounced and aggressive symptoms—frequent urination, pain, and pyuria—while tubercular disease begins slowly and insidiously. Hæmaturia rarely accompanies cystitis, but is a constant symptom in tuberculosis. Cystitis can usually be traced to

some definite cause, traumatic or otherwise, while tuberculosis comes on stealthily and without apparent cause: the urine from the tuberculous kidney will contain the characteristic bacilli until pyuria becomes permanent, and of course the presence of the bacillus renders it certain that tuberculosis exists somewhere along the genito-urinary tract, presumably in the bladder if other symptoms point in this direction.

Vesical calculus is accompanied by pain entirely different from that of tuberculosis—namely, a sharp, stinging pain, with intense desire to urinate; it is also attended by hæmaturia more constant than that of tuberculosis and more apt to follow exercise. Moreover, the pain and hæmaturia of stone are relieved by rest and the recumbent position, which make no difference to the symptoms of tuberculosis. Certain neuroses of the bladder might be mistaken for tuberculosis, but the absence of tuberculosis elsewhere, together with the absence of pain and hæmaturia, will differentiate one from the other. Tuberculosis of the kidneys cannot always be distinguished from tuberculosis of the bladder in the early stage; in fact, they are often associated, and it is always well to look for the disease in both places if it is found in either. But with tuberculosis of the kidneys there is almost always albuminuria and tube casts, these showing that the kidney is certainly inflamed; and if along with this there are reasons for suspecting tuberculous disease, the diagnosis may be regarded as wellnigh established.

The cystoscope is of great value in establishing the early diagnosis of tuberculosis of the bladder.

Prognosis.—While tuberculosis of the bladder is not curable either by internal remedies or surgery, its progress is so slow that the danger may generally be regarded as remote. The hemorrhage is generally slight, the pain not great, and the patient does not "wear out" rapidly, as is the case with cancer or sarcoma of the bladder. Some cases seem to advance only at long intervals, so that the patient enjoys periods of almost perfect health, and flatters himself that he has fully recovered. Yet it is doubtful if veritable tuberculosis of the bladder ever recovers.

Treatment.—So far as possible both physician and surgeon should let a tuberculous bladder alone. Irrigation is of no avail unless suppurative cystitis sets in, while it may be the means of inducing cystitis. No operative procedure promises any beneficial results; in fact, in view of the slow progress which the disease usually makes when let alone, and the ease with which cystitis is induced by meddlesomeness, the maxim should be to abstain entirely from local treatment until it is imperatively demanded on account of retention or sepsis.

The medical treatment of cystic tuberculosis is altogether "expectant." Pain may require anodynes, other symptoms may demand appropriate remedies, but no medication of any value can be addressed to the tuberculous bladder. All stimulating remedies, like sandal oil, copaiba, juniper, etc., should be studiously avoided. They are pretty certain to irritate the urinary tract, but they can do no good. Cod-liver oil, malt, guaiacol, creasote, or its carbonate may be prescribed for their tonic and antiseptic effects, but they will produce no specific or curative effect on the tuberculous bladder. The same may be said of tuberculin, the toxins, and other mysterious specifics from which we hoped so much, but have realized so little.

Defects of Development and Malpositions of the Bladder.

(*a*) **Absence of the Bladder.**—A few cases of this are on record. The ureters open by the side of or through the umbilicus, into the urethra, or into the rectum. A typical case is recorded in the *Lancet* for December 6, 1879, the ureters opening into the urethra, one of them being dilated and thrown into coils "like a child's intestine." Where the ureters become so dilated they probably act, as Mr. Reginald Harrison observes, as "subsidiary bladders."

In view of its extreme rarity, the improbability of its detection during life, and the impossibility of doing anything for its relief except to provide the patient with a urinal which can be attached to his person, absence of the bladder has no practical interest for the physician or student.

(*b*) **Two-cavity bladders**, double bladder or bladders divided by a median septum, have been seen by several observers. A. P. Smith of Baltimore met with a case of double penis with bladders to correspond; in one of the bladders he discovered a stone which was successfully removed, the patient making a good recovery.[1] Henry Morris quotes a remarkable case where a woman had "five bladders, five kidneys, and six ureters." "Four of the ureters emptied each into separate bladders, the other two into the largest bladder."[2] Such cases are interesting only as curiosities, so far as the physician is concerned.

(*c*) **Exstrophy or Extroversion of the Bladder.**—"A congenital deformity in which the symphysis pubis is absent and there is deficiency of the anterior wall of the abdomen and the bladder, with protrusion of the mucous surface of the latter through the opening" (Alexander Duane). This distressing condition is undoubtedly due to arrest of development. It is often associated with club-foot, hare-lip, cleft palate, spina bifida, and other congenital deformities the result of developmental inadequacy. It is very much more frequent in boys than in girls (in the proportion of 8.5 males to 1 female).

The posterior-wall of the bladder appears as a florid mucous disk, from the size of a nickel coin in children to that of a small sauce-plate in the adult. It occupies the space between where the symphysis pubis ought to be (but is not) and the umbilicus, or very near thereto in the fully developed adult.

It bleeds readily, is always wet with urine, more especially at its lower part, and is frequently painful. Near the lower margin are two small round or sometimes linear projections which mark the orifices of the ureters; by watching them one may see the urine escaping in feeble, intermitting jets. In one of my cases these jets were projected with considerable force. The ectopion or posterior segment of the bladder, thus pushed through the anterior abdominal wall, becomes continuous with the integument, so as to form a portion of the front wall of the abdomen. Below it is continuous with the rudimentary penis, upon the dorsum of which runs a gutter which represents the urethra. In the female there is a division of the labiæ and of the two sides of the clitoris.

[1] *International Cyclopædia of Surgery*, vol. vi. p. 335.
[2] *Diseases of the Genito-urinary Organs*, p. 327.

So far as the physician is concerned, the treatment is limited to the adjustment of some receptacle for the urine, so that it shall not continually dribble down, wet the patient's clothing, and make him repulsive to every one around him. Various operations have been devised with the object of relieving or curing this lamentable deformity, but so far with very poor success.

(*d*) **Herniæ Vesicæ, or Cystocele.**—"In the male, cystocele most frequently occurs in the inguinal canal; it may be either complete or incomplete, or may even extend, like an intestinal hernia, into the scrotum" (Reginald Harrison). It is, however, very rare in the male, and can only occur when the bladder is very much thinned and dilated and the inguinal canal in a state of very unusual patency.

Hernia of the bladder is not uncommon in females who are obese and loose-textured, who have borne children rapidly, and who are much of the time on their feet. It is very likely to be accompanied by obstinate constipation, hemorrhoids, and prolapsus uteri. The hernial pouch sometimes protrudes from the ostium vaginæ, and forms an elastic, rounded, fluctuating mass from the size of a pigeon's egg to that of an orange or even larger. In extreme cases the greater portion of the bladder may be involved. The cystocele acts as a diverticulum for the urine, which is retained for a long time, and then becomes decomposed and offensive. Cystitis finally invades the pouch, which is now occupied by a horribly offensive mixture of ammoniacal urine, muco-pus, and triple phosphates.

Other varieties of cystocele are mentioned. "The inguinal, femoral, obturator, and ischiatic foramina have all been the site of cystoceles accompanied or unaccompanied by a portion of bowel or omentum" (H. Morris).

The SYMPTOMS of vaginal cystocele are easily recognized. A rounded, elastic, fluctuating tumor, projecting into the vagina and protruding therefrom when the patient stands, which is not tender to the touch nor painful, which disappears more or less perfectly when the patient lies down, into which a bougie readily enters when introduced into the bladder through the urethra, and which can be emptied of its contents by introducing a catheter into the bladder and compressing the tumor with the fingers, can scarcely be anything but a cystocele. In spite of these perfectly obvious and indisputable pathognomonic symptoms, however, cases of cystocele have been mistaken for abscesses and opened for the purpose of evacuating pus. Let the physician simply introduce a catheter into the bladder, and his supposed abscess will begin to disappear at once.

Inguinal cystocele may be mistaken for, or may be accompanied by, intestinal hernia. The DIAGNOSIS is not always easy, but a cystocele is not resonant and gurgling like an intestinal hernia, nor is it doughy or boggy like a mass of omentum; it lessens in size and tension after urinating, and becomes swollen and tense if the urine is long retained.

Let the patient be required to lie in bed a few hours and retain his urine: as the bladder fills and becomes distended the cystocele will appear, and gradually increase in size until an elastic rounded tumor appears at the inguinal ring which is neither boggy nor resonant, and does not "gurgle" when compressed; now let a catheter be introduced

into the bladder, and the tumor will gradually subside as the urine flows out; this method will almost certainly establish a diagnosis. The same procedure can be applied to other erratic forms of cystocele if a tumor can be recognized by digital examination.

TREATMENT.—The treatment is mainly surgical. The hernial pouch should be kept empty if possible, and a truss or pad may be applied, which will alleviate the inconvenience and prevent increased protrusion. But the question of radical cure must be referred to the surgeon or gynæcologist.

(*e*) **Prolapse of the vesical mucous membrane**, or inversion of the bladder through the urethro-vesical orifice, sometimes occurs in women when the membrane is very flaccid and the urethral orifice has lost its normal tonicity. In some cases strychnine or ergot, with the local application of astringents, may do good, but generally surgical means, such as the actual cautery to the vesical orifice of the urethra, will be required.

(*f*) **Patent Urachus.**—Sometimes the upper segment of the intra-abdominal portion of the allantois fails to undergo closure and obliteration, as it should at about the seventh month of intra-uterine life, and then we have a "patent urachus," or a communication between the umbilicus and the bladder through which the urine may escape when the bladder becomes filled, and in which urinary concretions may form. It is frequently associated with some hindrance to free micturition, such as contracted meatus or phimosis.

TREATMENT.—The treatment consists in removing any obstacle to micturition which may exist, and closing the umbilical opening with a pad or by means of the cautery or sutures.

NEUROSES OF THE BLADDER.

Spasm.—Spasm of the bladder independent of inflammation or some other inherent cause is rare. It is sometimes met with in acute prostatitis, owing to the hypersensitiveness of the prostatic urethra, which causes reflex contraction of the detrusor urinæ, with forcible ejection of the urine. Hysteria is often accompanied or ushered in by a violent contraction of the bladder and the involuntary discharge of urine. Intense mental excitement, as a fit of anger, the "stage fright" of an amateur orator, or a sudden terror, will bring on vesical spasm and its unpleasant results. Of course in a given case it would be necessary to differentiate spasm of the detrusor from paralysis of the sphincter, both of which would be followed by involuntary urination. But in the former there would be the sensation of constriction and colicky pain in the bladder, accompanied by the forcible extrusion of the urine, while in the latter the pain would be absent and the escape of urine would be accomplished slowly and without force.

In the TREATMENT of vesical spasm the cause should be carefully sought and removed if possible, after which the bladder will usually resume its normal behavior. In some cases the antispasmodics, as valerian, musk, or the bromides, may be required, or a few suppositories containing one grain of codeine and five grains of iodoform may be prescribed, one of which shall be employed per rectum at bedtime.

Paralysis of the Bladder.—The bladder is a hollow muscular organ, composed of two layers of longitudinal fibres of unstriped muscle, which extend completely around the bladder from their origin anteriorly at the symphysis pubis to their insertion in the prostate in the male or the vagina in the female; and of an oblique layer, thin and irregularly scattered over the body of the bladder, but having no definite points of origin. The two layers of longitudinal fibres are known as the detrusor urinæ muscle. Of course, being composed of non-striated muscular fibres, the detrusor is not under the control of the will, but belongs to the class known as "involuntary muscles."

A somewhat thicker layer of unstriped circular fibres have undeservedly acquired the name of the "sphincter vesicæ internus;" these fibres are in no physiologic sense a sphincter, but they simply form a funnel-shaped cavity which is the beginning of the urethra. The true sphincter vesicæ or sphincter urethræ is situated a little lower down—is composed of striated fibres arranged circularly around the neck of the bladder and urethral orifice, extending down the urethra as far as the bulbous portion, and also sending a band of longitudinal fibres along the base of the bladder until they are lost in the oblique fibres. It should be borne in mind that the true sphincter vesicæ is a striated muscle of definite outline and location, and, for obvious reasons, is placed under the control of the will whenever it becomes necessary or expedient for the will to exercise its authority.

The nerves concerned in the mechanism of micturition are—(1) the motor nerves of the sphincter vesicæ, derived from the anterior roots of the third and fourth sacral nerves; (2) the sensory nerves of the urethra, derived from the posterior roots of the sacral nerves; (3) fibres from the cerebrum through the spinal cord to the motor fibres of the sphincter urethræ; (4) the inhibitory nerves "concerned in the reflex inhibition of the sphincter urethræ" (Landois), pass downward through the cord in the third and fourth sacral nerves; (5) "sensory nerves pass from the bladder and urethra to the brain, but their course is not known. Some of the motor and sensory fibres lie for a part of their course in the sympathetic" (Landois and Stirling).

From these facts we are quite warranted in the conclusion that the bladder is partly under reflex and partly under voluntary control, the reflex centre being the lumbar enlargement of the spinal cord and the voluntary the cerebral cortex.

In the lumbar portion of the cord there are probably two reflex centres—one for the detrusor and the other for the sphincter (J. Tyson).

Under ordinary circumstances the urine is retained in the bladder by the reflex contraction of the sphincter, but as the organ becomes distended the fibres of the detrusor show a tendency to contract and expel the urine, whereupon the power of the will, acting through the voluntary centre, is brought to bear upon the sphincter, which now resists the efforts of the *detrusor*, and the urine is retained until the bladder is fully distended or until it can conveniently be relieved by micturition.

Of course there may come a time when the distention of the bladder is so great as to provoke the detrusor to override the control of the will over the sphincter, and the organ is emptied involuntarily.

Paralysis of the bladder may involve the detrusor muscle alone, as

happens in lesions of the cord involving the motor tracts, in which case there will be complete retention unless communication with the brain remains intact, when an act of the will may suspend contraction of the sphincter, and cause pressure on the bladder by contraction of the abdominal muscles, with partial emptying of the organ. All degenerative lesions of the motor column of the spinal cord are attended by progressive paralysis of the detrusor and more or less complete retention of the urine. As the degeneration of the cord progresses, the sensory columns are generally implicated, the sphincter vesicæ loses its power of contraction, and the urine dribbles away without the patient's knowledge or power to control it. Under these conditions the bladder may be distended with urine, in spite of the fact that it continually escapes—a fact which the physician must not forget in the care of such patients.

Again, the sphincter may alone be paralyzed, when there will be continual incontinence, owing to the fact that the detrusor remains in action. Coughing, sneezing, or laughing will produce a sudden gush of urine, the sphincter being unable to resist the outflow.

Paralysis of the sphincter is always more or less complete in apoplexy, and is a constant concomitant of degenerative changes of the brain. In fact, the rapidity of invasion and extent of the loss of power in the sphincter is not a bad index of the progress of disease in the brain. So long, however, as the patient retains his power over the abdominal muscles he may partially empty the bladder by "straining" or voluntarily contracting the abdominal muscles and thus compressing the bladder.

TREATMENT.—The treatment of bladder paralysis depends upon the variety and cause. Cases which depend upon lesions of the nervous centres are of course incurable, since the cause cannot be removed. Strychnine and electricity may produce temporary benefit, but the progress of the disease of the nerve centre will inevitably cause the bladder paralysis to advance in spite of all treatment.

Retention which is due to spasm of the sphincter must be cured by ascertaining and removing the cause. Such palliative remedies as belladonna or the bromides, or those combined, will be found useful and sometimes curative. Some cases will require circumcision, some will depend upon filth balanitis, some upon rectal irritation as from pinworms, and some cases in very young girls will be cured by treating vulvitis or vaginitis and the administration of a timely lecture on cleanliness to a delinquent mother. Nearly all cases of bladder spasm depend upon some ascertainable and removable cause—a fact which we are prone to forget in our clinical work.

Retention of urine dependent upon paralysis must be relieved by the catheter, and in many cases it will be necessary to teach the patient or an intelligent nurse how to pass the catheter. Great pains must be taken to render the catheter aseptic, as neglect of this important matter will certainly induce septic cystitis. Sooner or later nearly all cases become septic, and then irrigation of the bladder will be necessary.

Specific directions as to methods and remedies will be found in the article on Chronic Cystitis (p. 825).

Incontinence of Children.

Well-trained children who are in health learn to use the commode in urinating from the age of eighteen months to two years. Children who are neglected or are allowed to "come up themselves" will sometimes wet their clothing by day and their beds at night till they are five or six years old. Incontinence may therefore cease in one child before he is two years old, while it may last in another till he is six years old.

Nocturnal incontinence of course lasts longer than incontinence during the daytime, since during sleep the will ceases its watchful care over the sphincter.

Some children lose control over the sphincter after acquiring it, but this generally occurs before they are six years old.

In some cases the incontinence occurs only at night; in others only during the day, while still others have incontinence both day and night. But in the great majority of cases it takes place only at night.

Many cases of daytime incontinence are the result of carelessness or thoughtlessness. The child is deeply absorbed in his play, and does not heed the call to urinate until he is surprised by the unexpected passage of urine into his clothes. Such cases are easily cured by watching and timely admonition.

Children who have nocturnal incontinence are generally profound sleepers, or else they belong to the pale, anæmic, nervous class, and are poor sleepers.

In the first class there is generally no essential cause for wetting the bed except the deep unconsciousness of sound and perfect sleep in a hearty, vigorous child. Such children should be awakened and made to urinate late at night, so as to avoid distention of the bladder during the early morning hours.

In the second class there is an "irritable" bladder, which always implies an excitable detrusor muscle ready to contract on the smallest provocation; a weak and unstable nervous system, which never finds rest, even in the hours of darkness; the child is wakeful and peevish, often starts from sleep in fright or terror, and frequently has involuntary urination by day or night, but certainly at night. These are the so called "neurotic" cases of incontinence.

There are also a group of cases occurring in dull, slow-thinking children anywhere from six to ten years of age, who do not seem to care whether they urinate in one place or another. During the day they have no sense of modesty or shame, and they wet the bed night after night with no feelings of mortification or sorrow. I knew such a case about fifteen years ago, but, to the surprise of every one, that dull and stupid boy has developed into a smart and energetic business man.

I have also met with several cases of nocturnal incontinence in bright, intelligent girls from ten to fifteen years of age which have proved very obstinate. These patients have been mostly of the "precocious" kind, who have been unwisely indulged as to food, society, and amusements, and whose nervous systems were consequently on a "wire-edge" of excitement, which precluded healthy, restful sleep and induced "irritability" of the genito-urinary centres.

ETIOLOGY.—The causes of incontinence frequently elude our most careful research. In only a small minority of cases can a well-defined cause be discovered. E. M. Buckingham well says: "A study both of the literature and our individual patients should convince us that we are not dealing with one condition, but with a symptom common to many conditions."[1]

The reflex irritation produced by pin-worms, obstinate constipation, phimosis, balanitis, vaginitis and vulvitis, vesical calculus, and allied sources of bladder excitation may result in incontinence, especially at night, but I have examined more cases for which I could assign no adequate cause than cases for which a reasonable cause could be found. It is certain that urine loaded with lithic acid or oxalates will produce sufficient bladder irritation to cause nocturnal incontinence; albuminuria is said to be a cause, but I have never seen a case produced thereby; diabetes is of course more than likely to be accompanied by incontinence, owing to the nocturnal distention of the bladder; epilepsy and incontinence frequently go together, even without nocturnal epileptic seizure. E. M. Buckingham[2] holds anæmia responsible for many cases —an opinion with which I fully agree. The same writer also calls attention to the fact that "incontinence is far more troublesome in winter than in summer."

TREATMENT.—The treatment of incontinence is sometimes followed by a prompt and permanent cure, but more frequently the results are disappointing and unsatisfactory.

In view of the fact that the efficient cause is so likely to be a matter of guesswork, it follows that in many cases the treatment must also be a matter of guesswork, or, in other words, empirical.

When the cause can be discovered its prompt removal should precede all other treatment. Pin-worms, constipation, phimosis, vesical calculus, and allied causes should be sought for and removed if present. In some precocious children, both boys and girls, who have not had proper maternal care, self-abuse will be found responsible for a wet bed. I have seen many cases promptly cured by evicting a colony of pin-worms from the rectum and thoroughly unloading the colon of its impacted fæces. The parents must see to it that the child's habits are correct, as regards eating, drinking, defecating, and urinating. The child should sleep in a well-aired room, on a moderately hard bed, and he should go to bed at a seasonable and regular hour. He should not be allowed to drink intemperately of water or other liquids before retiring; and he should be awakened as late as eleven o'clock and be required to empty the bladder. Elevating the foot of the bed so as to keep the urine from coming in contact with the trigonum vesicæ has been recommended.

Incontinent children should take light suppers, or rather suppers of plain light food in moderate quantity; they should be taught to eat slowly and not to "bolt" their food unmasticated, as so many children, even in "polite" families, are in the habit of doing. Sweetmeats, highly seasoned food, and candy should be forbidden, especially at the evening meal. The object of all this is to send the child to bed with the stomach empty, or as nearly so as possible, so that there shall be no

[1] *American Text-book of Diseases of Children*, p. 968. [2] *Op. cit.*, p. 969.

distention from flatulency, which will distend the stomach and bowels and cause them to compress the bladder, and provoke it to contract and expel its contents while the child is asleep.

Anæmic incontinence will require iron, malt, or cod-liver oil, and many cases will recover without other treatment.

Faradization with one pole over the lumbar region and the other in the urethra or vagina has been recommended, but there are obvious objections to this practice, especially with girls.

Massage has its advocates, and good results have been reached thereby, the proportion of cures and failures being about evenly divided. Sawyer advises massage of the sphincter by "introducing a probe into the bladder and exercising gentle pressure backward and from side to side"—a practice which I would neither adopt nor recommend.

The examination of the urine will frequently give the physician valuable suggestions as regards treatment. Lithuria will call for potassic citrate, dissolved in some demulcent; glycosuria will require antidiabetic regimen and treatment. It is scarcely necessary to say that the urine should be examined in all cases before treatment is commenced.

Belladonna has long been recognized as one of the most valuable remedies in nocturnal incontinence, and especially in cases depending upon irritability of the detrusor. It is usually necessary to give it in ascending doses until the pupil shows the characteristic dilatation. It is altogether best to give the entire daily quantity after four o'clock in the afternoon—say one dose at four or five o'clock, and another at eight or nine in the evening, so as to have the most marked effect during the "small hours" of the morning when sleep is most profound and the sphincter is most likely to be taken by surprise. Cases which depend upon atony of the sphincter are not benefited by belladonna; in fact, they are apt to be made worse; yet it is a fact that is borne out by clinical experience that belladonna relieves more cases of incontinence than any other remedy.

The bromides of potassium and sodium are also very useful in similar cases. They must be given in full doses, and the last and largest dose should be given at bedtime. Good results are sometimes obtained by combining belladonna with the bromides.

Chloral hydrate, cannabis indica, and other narcotics have been recommended, but they are next to useless so far as incontinence is concerned, and there is always some danger of the formation of a habit which is hard to break off. Rhus aromatica is favorably mentioned by W. Whitla, but I have never tried it.

When incontinence depends upon atony of the sphincter, strychnine is the remedy *par excellence*. It must be given in ascending doses until definite results are reached. In a case of uncomplicated atony or weakness of the sphincter a cure is wellnigh certain if strychnine is given continuously and in full doses. Of course if the case be one of paralysis of the sphincter depending upon some organic disease, neither strychnine nor anything else will cure it.

In conclusion, I wish to enter an emphatic protest against attempting to cure a child of wetting the bed by punishment. It is useless, senseless, and inhuman. It never cures incontinence, but it does throw a sensitive child into a state of high-strung, nervous excitement, which

predisposes him to do just what the brutal castigation is expected to cure.

Trichiasis Vesicæ.

Hairs are occasionally passed with the urine. Lebert states that the hairs which are met with in the urinary passages "may either have developed there, or have sprung from dermoid cysts which have opened into the bladder, or may have been introduced from without."[1] Nevertheless, I can find no recorded case in which hairs have actually grown from the vesical mucous membrane, and I do not believe such a case ever has occurred.

To quote Lebert again:[2] "It is very uncertain and improbable that there is such a condition as idiopathic trichiasis vesicæ."

Masses of hair, portions of skin, bones, teeth, and other fœtal relics have been found in and removed from the bladder, both ante- and post-mortem, having been derived from dermoid cysts which have ruptured and discharged their contents into the bladder. Mr. Joseph Thompson removed from the bladder of a woman by operation various portions of a fœtus.[3]

Carlson records a case in which after death a stone "as large as a goose's egg was found in the bladder, from parts of which hairs had grown out."[4] By most of us, however, this opinion, so far as it relates to the growth of the hairs, will be received *cum grano salis*.

Hairs are sometimes thrust into the bladder by hysterical females, along with goose-quills, hair-pins, and other equally bizarre materials, but the goose-quills and the hair-pins are quite as likely to *grow* in the bladder as are the hairs themselves. The hirsute contents of the human bladder always get there from without the organ, and not infrequently from without the body.

The treatment of trichiasis vesicæ consists of simply removing the hair from the bladder and the subsequent treatment of the cystitis which is usually present. The hair and other foreign bodies can generally be taken out through the urethra, since these cases almost always occur in females, although dermoid cysts are, on rare occasions, found in men. Most patients who are troubled with hairy bladders will be benefited by a little moral treatment and an accretion of common sense.

PROSTATITIS.

(A) Acute Prostatitis.

The causes of acute prostatitis, according to Henry Morris, are inflammation from neighboring organs, direct irritation like caustics to the urethra, cantharides or other irritant drugs, calculus or foreign bodies, constipation, lewdness, and sitting on damp grass or cold stone or iron seats. Of course venereal infection comes in for a large share of the blame, and in this category we reckon acute urethritis, gleet, and stricture. It may occur at any age above eighteen or twenty, but is most likely to occur between twenty-five and forty.

[1] *Op. cit.* [2] *V. Ziemssen*, 731. [3] *Lancet*, 1863, vol. ii. p. 621.
[4] *Diseases of the Bladder*, p. 226.

PATHOLOGICAL ANATOMY.—The gland is much swollen and turgid with blood; the prostatic urethra is reddened and swollen; at a later stage pockets of pus are found occupying the ducts of the gland. The ejaculatory ducts are distended with pus and sometimes ulcerated.

SYMPTOMS.—A sense of aching and heaviness in the perineum is the first indication of prostatitis; the pain is sometimes severe and of a throbbing character. Frequent micturition, often of an urgent, imperative type, soon comes on, and the act of urination is sometimes intensely painful, especially when the bladder and adjacent muscles contract toward the close of urination. There is rectal pain, later rectal tenesmus; defecation produces great suffering, and frequently an acute attack of hemorrhoids may develop. Digital examination of the prostate reveals a swollen, hot, and tender prostate, and the whole rectum is hot, tender, and swollen.

DIAGNOSIS.—The diagnosis is very easy, the only question being between prostatitis and periprostatitis, which will be described presently. Digital exploration of the rectum will at once reveal the large swollen, tender prostate, and the diagnosis will be established.

PROGNOSIS.—Prostatitis usually ends in recovery, either as a result of resolution without suppuration, or suppuration with spontaneous discharge into the urethra or rectum, or evacuation after incision. A case which is not diagnosticated or properly treated may result in chronic prostatitis with hypertrophy, but the clinical fact is that few cases of prostatic hypertrophy can be traced to acute prostatitis as a cause. Neglected or unrecognized cases are far more likely to result in troublesome fistulæ which burrow into the perineum and require surgical treatment for their relief.

TREATMENT.—Rest in bed, leeches, followed by hot fomentations to the perineum, with an occasional hot sitz-bath; rectal douches of water as hot as can be borne, given two or three times a day and retained for half an hour; morphine or codeine in full doses per rectum sufficiently often to relieve pain and tenesmus, with liquid diet, will answer every indication unless suppuration occurs, when the case must be transferred to the care of a surgeon.

Periprostatitis or periprostatic abscess sometimes occurs in "the cellular tissues of the space between the rectum and prostate and the deep triangular ligament of the perineum" (Morris). It is generally a sequel or accompaniment of prostatitis, but occasionally occurs without involving the prostate.

The SYMPTOMS are very similar to those of prostatitis, except that the rectal and perineal pain and throbbing are greater and the area of tenderness and fluctuation is more extended.

The TREATMENT is precisely the same as for prostatitis, but the services of a surgeon are quite certain to be required, since resolution is very unlikely to occur.

(B) CHRONIC PROSTATITIS.

Chronic prostatitis may follow an acute attack or the latter may merge into the former, but this does not often happen.

Its most common cause is certainly gonorrhœa, the specific germs

(gonococci) finding their way into the follicles and there setting up an inflammatory process. But it may be caused by cold and wet, long-standing stricture, vesical calculus, and venereal excess. It is most likely to occur between the ages of twenty-five and forty-five years, and must not be confounded with senile hypertrophy of the prostate, which is met with in men from sixty-five years upward.

PATHOLOGICAL ANATOMY.—The gland is not much enlarged, but is rather soft, elastic, and spongy. Small pus pockets with colonies of gonococci are found scattered about the organ. The prostatic urethra is reddened, swollen, and sometimes ulcerated, and the prostatic ducts are dilated and filled with a dirty, thick muco-pus. In rare cases a single prostatic abscess of considerable size may form.

SYMPTOMS.—Rather frequent micturition, with muco-purulent discharge, perhaps containing a few drops of blood; a feeling of heaviness or fulness, with some perineal pain, which is increased by exercise, especially horseback riding or bicycling; tenderness of the prostate on digital examination, the gland feeling rather soft, elastic, and doughy, together with pain in the prostatic urethra on passing a sound or catheter, which is generally followed by a few drops of blood. If the urine is passed in two portions, the portion first voided will contain muco-pus or blood, or both, while the last portion will be nearly natural in appearance.

The DIAGNOSIS presents no difficulties if a little care and caution is used. Some cases produce symptoms very much resembling vesical calculus, but the sound will soon decide the question by eliciting the characteristic tenderness in the prostatic urethra and failing to detect the suspected calculus.

TREATMENT.—Rest, perineal counter-irritation in the form of small blisters, iodine, croton oil, or ointment of red iodide of mercury, care being taken that the irritant agent does not extend to the anus or scrotum; suppositories of iodoform or ichthyol, combined with codeine if necessary, and a daily douche of hot water,—will be the most efficient means of treatment.

Morris recommends the instillation of silver nitrate (5 grains to the ounce) or chloride of zinc (1 grain to the ounce) into the prostatic urethra if the above measures do not work a cure in five or six weeks. I have found hot fomentations to the perineum useful. Of course if an abscess in or around the prostate forms, it must be evacuated at once.

As these cases generally occur in debilitated nervous people, general tonics will be required, such as iron, quinine, malt, and phosphatic preparations. A change of climate, especially a visit to the seaside, with sea-bathing, sometimes works wonders.

(C) PROSTATORRHŒA.

In connection with chronic prostatitis, or it may be without it, we meet with cases in which there is a thick, ropy discharge from the urethra, especially in constipated patients. This is known as "prostatorrhœa," and depends on a catarrhal inflammation of the prostatic follicles with thick, sticky secretion. It is frequently induced by sexual excess, and is generally a source of much solicitude and despondency

on the part of its victims, who see impotency and "lost manhood" looming up in the distance. A microscopic examination of the discharge will eliminate suppurative prostatitis by showing the absence of pus, and spermatorrhœa by showing the absence of spermatozoa. The simple and comparatively harmless nature of the complaint will thus be established.

The TREATMENT is chiefly moral: the patient should be assured that his trouble involves no danger; constipation should be relieved by appropriate remedies, whereof aloes should *not* be one; tonics should be given if required; but, above all, the patient's mind should be diverted into a healthful channel, so that his thoughts and aspirations shall tend upward and away from the lower animal propensities. A course of mental gymnastics is the best substitute for venereal follies and "lost manhood."

(D) Senile Hypertrophy of the Prostate.

Senile hypertrophy of the prostate with cystic induration, being an essentially surgical disease and requiring essentially surgical treatment, is not discussed at length in this work. Its consequences—namely, chronic cystitis with dysuria—have already been treated in detail in the foregoing pages (p. 822 et seq.).

ABNORMALITIES OF THE URINE.

By THOMAS D. COLEMAN, M. D.

General Considerations.—The kidneys, ureters, urinary bladder, and urethra comprise the urinary organs. All of them are concerned in the elimination of urine from the body, but the kidneys only take part in the excretion of urine, while the ureters and urethra serve as conducting passages, and the bladder functions as an organ of convenience or reservoir to collect and hold the urine until such time as it may be convenient or necessary to void it. Normally, the ureters, bladder, and urethra simply add to the urine a small quantity of mucus, but beyond this they do not cause any change in it. When one considers that the fæces consist of the indigestible residue of food taken, and that little if anything is added to this residue by the tissues of the body, and that the chief carbon waste is accounted for in the carbonic acid exhaled from the lungs, and that practically all of the products of destructive nitrogenous metabolism in the body are found in the urine, the importance of this fluid excretion as an index of the general condition of the body can scarcely be overestimated.

Its importance is shown by the fact that no serious disturbance of any portion of the organism can develop without change in the normal constitution of the urine taking place, and such change (most often) as to give a valuable index of the nature of the trouble. Effete matters when produced by normal processes are injurious to the organism; then how much more so must they be when they are secreted in excessive amounts by morbid processes! The advances that are being made in knowledge of diseased conditions, serve to emphasize the value of an accurate examination of the urine in making a diagnosis. Such an examination will often enable one to diagnosticate a diseased state when no other sign or symptom is present. The kidneys are therefore the chief and most important organs of excretion for the body, serving as they do to separate from the blood water, salts, and the other waste products resulting from nitrogenous metabolism.

Functional activity on the part of the kidneys results in the formation of urine, and its composition varies, even in health, within tolerably wide limits. There is a relative difference in degree in the normal effete excretions of the child with its active tissue construction and destruction, and in the aged when the tissues have passed life's meridian and less active retrograde changes are going on. There is also some variation due to the seasons of the year, and there is, too, a fairly constant diurnal variation. Again, it is plain that the normal waste products will appear in the urine temporarily in excess following the ingestion of food

and times of day when the body is in a state of activity; and that, on the other hand, they will be temporarily diminished following periods of fasting and times when the body is at rest.

When, therefore, tissue metabolism throughout the body is normal, and when the kidneys perform their excretory functions in a healthy manner, the urine will be normal; and, on the other hand, when one or both of these are impaired or perverted in action this fluid will suffer change that may be considered abnormal or anomalous. The normal constitution of the urine, then, may be affected in either of two ways—viz. one or more of the tissues of the body may fail in the performance of work or imperfectly function, and in consequence the blood becomes less surcharged with effete matter, and so the urine is deficient in it; or waste products may be present in the blood in normal quantities, but the kidneys through disease may not be able to remove them, and the urine will similarly exhibit change. Either of these conditions may produce embarrassment to a part of the body or the organism as a whole. It will be seen, therefore, that the composition of the urine must give an index of the character, and not infrequently the degree, of tissue change going on in the body, and also that a careful examination of this fluid is frequently the only means by which a diagnosis of diseased conditions can be made with any degree of accuracy.

Since normal ingredients when present in greatly increased or diminished quantity constitute an abnormal or unhealthy condition, it will be convenient in what is to follow to consider the conditions together, showing where the normal merges into the abnormal, and finally to consider those constituents that may at all times be regarded as abnormal.

Quantity.—The average quantity of urine excreted by a healthy adult in twenty-four hours amounts to between 1200 and 1500 cubic centimetres (nearly 3 pints), and varies, even normally, within fairly wide limits. The quantity excreted will vary with the quantity of fluid taken, with the activity of excretion on the part of the skin and lungs, with the blood pressure, and with the general condition of the tissues. There may be a temporary increase, caused (*a*) by the drinking of large quantities of water; (*b*) by chilling the surface of the body, thus diminishing the calibre of the skin capillaries and lessening, for a time, the activity of the sweat glands; (*c*) by exercise, nervous excitement; and (*d*) by the action of various drugs belonging to the class of diuretics. The quantity may be increased pathologically in such diseases as diabetes mellitus, diabetes insipidus, in chronic nephritis, especially when granular or contracted kidneys exist; in hysteria, convulsive seizures, etc.

Temporary diminution of the quantity of urine normally excreted may be found on curtailing the amount of fluid usually ingested in the twenty-four hours or entirely withdrawing it; after profuse sweating and excessive activity on the part of lungs; by diminished blood pressure and by the action of certain drugs. The quantity may be diminished pathologically in acute general inflammatory diseases and in fevers; in the first stage of acute nephritis and in the exacerbations of the chronic form; in organic diseases of the heart resulting in cardiac insufficiency, and in disorders of the liver inducing dropsical effusions; by mechanical obstruction to the exit of the urine—*e. g.* by tumors, calculi, etc.—and after dysenteric attacks and copious diarrhœal

discharges. In cholera the urine is much diminished, and toward the close, the choleraic discharges may be so great as to cause entire suppression of the urine.

Anuria, or suppression of the urine, may be produced by plugging up both ureters by calculi or pressure on them by adventitious growths, by exhausting hemorrhage, cholera, acute and chronic nephritis, yellow fever, and by excessive diarrhœal and dysenteric discharges. It is desirable at all times to know the quantity of urine that the patient is excreting, and for analytical purposes it is best to take a portion of the mixed urine that has been passed in twenty-four hours.

Color.—The color, like the quantity, is subject to variation. The amount of pigment contained in normal urine, being practically constant, it will impart less color to the urine when the quantity excreted is large, and *vice versâ*. It is usually of a light, straw, amber, or reddish color, and varies with the degree of concentration. The color will be increased normally after excessive sweating, after withdrawal of or diminishing the quantity of drinking water, and when the quantity of urine excreted is lessened from any cause; it will be diminished by drinking large quantities of water, by nervous excitement, and by convulsive and hysterical seizures. Diuretics as a class diminish the color, because by heightening the arterial tension they increase the relative proportion of water; and certain drugs intensify and even impart characteristic colors to the urine—*e. g.* rhubarb produces a gamboge yellow; senna, a brown; naphthaline, carbolic acid, and creasote, a green or even black color.

The color may be increased pathologically in inflammatory disorders, in fevers, in renal congestion, in hepatic diseases which cause to be formed and pass into the urine an excessive amount of bile pigment, in hæmaturia and in hæmoglobinuria, in diarrhœa, entero-colitis, etc. The presence of chyle will cause the urine to have a milky appearance, and the color is diminished in diabetes mellitus, diabetes insipidus, chronic nephritis, and anæmia.

The color is an important feature, and, taken in connection with other clinical signs, is of service from a diagnostic point of view.

Odor.—The odor of normal urine is characteristic and may be described as urinous or aromatic. This may be changed in disease to an ammoniacal, and even a sweetish smell, as is found in diabetes mellitus. Various drugs impart characteristic odors to urine. Among them may be mentioned cubebs, copaiba, oil of yellow sandalwood, valerian, turpentine, carbolic acid, etc.

Taste.—Normal urine has a bitter, salt taste, but gyclosuric urine may have a sickening sweet taste.

Reaction.—The reaction of normal urine is acid, except after a generous meal, when it may be neutral or feebly alkaline. The acidity is due chiefly to the contained disodic phosphates, and in part also to uric acid, lactic acid, hippuric acid, oxalic acid, and their salts.

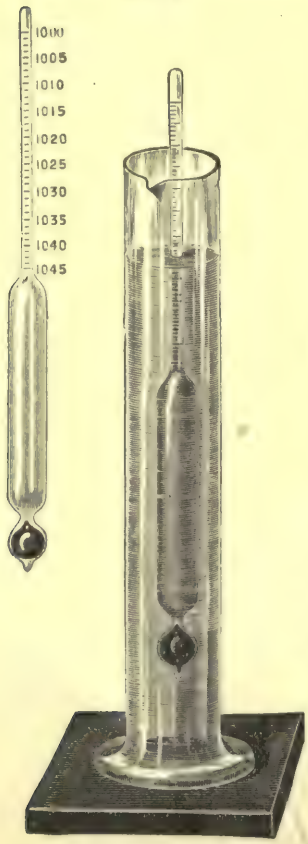
The reaction is ascertained usually by blue and red litmus paper, or a violet amphoteric paper may be used. If the urine be acid, a drop of it when placed on blue litmus paper will turn it red; if alkaline, the drop when placed on red litmus paper will turn it blue. If the alkalinity be due to ammonia, this will volatilize on allowing the litmus to dry

in the air and the blue spot will disappear; if it be due to a fixed alkali, the blue spot will remain. If the urine be perfectly neutral, it will not effect change of either the red or blue litmus. Solutions of litmus may be used instead of litmus paper, and in this way the degree of acidity or alkalinity may be ascertained. The urine of herbivorous animals is alkaline except when they are fasting, in which event they are practically carnivorous, since they are then living on their own flesh.

The acidity of human urine may be increased by starvation and after taking acids. It may also be increased in fevers, gout, rheumatism, and diabetes mellitus.

The normal acidity may be diminished, and even changed to alkaline, by an exclusive vegetable diet, by the ingestion of alkalies and the alkaline salts of acetic, citric, and tartaric acids. These last become converted in the system into carbonates. If the urine is alkaline when passed and the above abnormal conditions can be excluded, it indicates a catarrhal or purulent inflammation of the mucous membrane of the bladder or renal pelvis.

FIG. 54.



Urinometer (Charles).

Normal urine on standing in the air generally increases in acidity from an increase of phosphates, but this slowly disappears under the action of a ferment produced by a putrefactive micro-organism (*micrococcus ureæ*) and the fluid changes to alkaline. That this micro-organism enters by atmospheric contact is proven by the fact that if fresh urine be sealed up it will remain acid for many months. The ferment produced by the micrococcus ureæ decomposes the contained urea into carbonate of ammonia. When such decomposition takes place the urine becomes much more cloudy from the precipitation of the phosphates of lime, ammonio-magnesic phosphates, and urate of ammonia. Normal urine is naturally aseptic, but forms an excellent culture medium for micro-organisms, so that a cystitis may be easily set up by the introduction of infected instruments into the bladder.

Specific Gravity.—Taking distilled water as the standard, the specific gravity gives an index of the quantity of solids contained in the urine. Just as the quanti[illegible] color, and other normal ingredients are not absolutely fixed, so is t[illegible] specific gravity a somewhat variable quantity. It may range from [illegible]15 to 1025, and even slightly above and below these points tempo[illegible], without being regarded as abnormal. In health anything that [illegible]s to increase the quantity of urine excreted will in like manner co[illegible]tribute to lower the specific gravity,

and *vice versâ*. The specific gravity is determined by means of a urinometer which is known to be accurate. It may easily be tested by placing the instrument in distilled water at a given temperature, when, if the instrument is accurate, it should register 1000. To determine the specific gravity of a specimen of urine a cylindrical glass vessel should be used whose diameter is just sufficient to allow the urinometer to float freely in the liquid without touching the sides of the glass. Having filled this receptacle about two thirds full of urine, the urinometer is allowed to float freely in the contained fluid, and the record is taken at the point where the bottom of the meniscus touches the registered scale on the slender portion of the urinometer.

Since the specific gravity varies with the quantity of contained solids, it is essential that it shall be estimated from a portion of the entire amount excreted in twenty-four hours. A fairly accurate estimate of the contained solids may be obtained quickly in this way by employing Trapp's method, which consists roughly of doubling the last two figures of the specific gravity for every 1000 parts of urine, or, more accurately, of multiplying the last two figures of the specific gravity of a given specimen by 2.33. Thus, if the urine has a specific gravity of 1020, then $20 \times 2.33 = 46.60$. If the amount passed in twenty-four hours equals 1500 c.c. the amount may be calculated by proportion, as follows:

$$1000 : 1500 : : 46.60 : x = \frac{1500 \times 46.60.}{1000} = 69.90$$

It should be remembered that the only way to determine accurately the quantity of solids is to separate them for the entire twenty-four hours by complicated chemical processes and weigh them. The amount of solids excreted daily by a man of average size is about 2 ounces (70 grammes). Anything which tends to increase the watery constituents of the urine, unless the solids are proportionately augmented, will contribute to produce a low specific gravity. Thus the specific gravity will be diminished normally by drinking large quantities of water, chilling the surface of the body, nervous excitement, etc., and opposite conditions will increase the specific gravity.

Pathologically, the specific gravity is increased in fevers, in acute inflammatory disorders, in diabetes mellitus, etc. It is diminished in diabetes insipidus and in acute and chronic nephritis.

When the specific gravity does not rise and the quantity of urine is diminished, it indicates that either the metabolic processes resulting in the formation of urea, uric acid, etc. are interfered with, or that the kidneys are failing to perform their proper functions in removing these effete products from the blood. It is therefore an unfavorable sign near the termination of attacks of fever and nephritis to find that the quantity of urine remains the same, while the specific gravity diminishes. As already intimated, in the former case it indicates a depression of the metabolism normally going on in the tissues, and in the other an inability on the part of the kidneys to remove the results of tissue waste from the blood. V. Jaksch has called attention to the fall in density which may for several days precede uræmic convulsions, and empha-

sizes it as a most valuable clinical sign. When uræmia develops without lessening the quantity of urine passed, the specific gravity is invariably diminished. It will be seen, therefore, that the determination of the specific gravity is valuable from a clinical standpoint, and should be employed frequently as an aid to diagnosis.

Consistency.—Normal urine is quite limpid, like pure water, but it may become altered by the contained solid ingredients—*e. g.* much mucus will make it ropy, and excess of sugar will make it of syrup-like consistency. Ordinarily, too, the urine is quite clear. It may be clouded temporarily by excess of salts from a too generous indulgence in certain kinds of food and by mental exertion, or it may be clouded abnormally by various salts held in suspension, by blood, pus, epithelial débris, and mucus.

It is important in all cases to note the presence or absence of sediments. To this end it is well to put the urine in a conical glass, cover it to exclude atmospheric impurities, and allow it to remain for twenty-four hours, or, better still, to make use of the centrifuge. In this way the solid ingredients will collect at the bottom of the glass, when they may be examined chemically and microscopically. It should be borne in mind that the microscope will give the only accurate information concerning the composition of most sediments.

Composition.—The constituents of the urine may be divided into the inorganic and the organic. These occur in twenty-four hours, in a man of average weight of 66 kilos (145.55 pounds), in the following proportions:

Amounts of the Several Urinary Constituents passed in Twenty-four Hours by an Average Man of 66 Kilos (Parkes).

Inorganic.			*Organic.*		
Water	1500	grammes.	Urea	33.180	grammes.
Sulphuric acid	2.012	"	Uric acid	0.555	"
Phosphorus	3.164	"	Hippuric acid	0.400	"
Chlorine	7.000	"	Kreatinin	0.910	"
Ammonium	0.770	"	Pigment and other substances	10.300	"
Sodium	11.090	"			
Potassium	2.500	"			
Calcium	0.260	"			
Magnesium	0.207	"			

A perceptible and persistent increase or diminution of any of the above ingredients or the addition of other substances wholly foreign to normal urine may be regarded as an anomaly or an abnormal condition.

The water originates from the fluid contained in both the food and drink.

Chlorides.—Chloride of sodium is by far the most abundant inorganic constituent of the urine. The quantity excreted in twenty-four hours amounts to from 10 to 13 grammes. There are also present in the urine in small quantities the chlorides of potassium and ammonium, calcium, and magnesium. Chlorides are derived from the food, and may be detected by adding to urine, previously acidulated with nitric acid, a few drops of a dilute solution of nitrate of silver, when a dense white, flocculent precipitate of chloride of silver will form, which is not dissolved by excess of acid. They are diminished in quantity, and may almost

disappear when salt is withheld from the food and in fevers. In pneumonia chlorides are usually entirely absent. They appear again when the stage of resolution commences. As such disappearance is an aid to diagnosis, it is well to frequently examine the urine of patients suffering with pneumonia, and as a control experiment, to give some idea of the diminished quantity of chlorides, to use the same silver solution with a specimen of normal urine. Chlorides are also diminished in acute and chronic nephritis, roseola, variola, scarlatina, typhus fever, etc. In dropsy, when the fluid accumulation is rapidly developing, there is a diminution, but when the dropsical effusion is being carried off the chlorides are much increased. They are also increased in amount as convalescence from the above mentioned diseases is taking place.

Phosphates.—Phosphoric acid does not occur free in normal urine, but in the form of phosphates of sodium, potassium, calcium, and magnesium; and in urine which has undergone ammoniacal fermentation a double salt of ammonio-magnesic phosphate is formed.

The two varieties of phosphatic salts usually found are—(1) the earthy phosphates of calcium and magnesium, which are insoluble in water, being precipitated by alkalies, but dissolving on the addition of acid. These are found in alkaline urine. (2) The alkaline phosphates, which are composed chiefly of acid sodium phosphate, and in much less degree of potassium phosphate. These are found in acid or neutral urine. The latter class in large part constitute the bulk of phosphates of the urine. They are soluble in water, and are not precipitated on the addition of alkalies.

The acidity of normal urine is due almost entirely to the presence of acid sodium phosphate; uric, lactic, hippuric, oxalic, and other acids perhaps adding something thereto. Between 3 and 4 grammes (46 to 61.73 grains) of phosphates are excreted in twenty-four hours. A physiological increase in the phosphates occurs after eating certain kinds of foods and undue mental strain.

Phosphaturia signifies the occurrence of phosphates in persistently excessive quantities in the urine. The diagnosis should not be made without careful examination of all the phosphates excreted in twenty-four hours. The density of the urine, form of diet, and trivial disorders may frequently cause an apparent or merely temporary increase in the phosphates; hence unless the quantity excreted in twenty-four hours be determined, and for a period extending over several analyses, the decision would be untrustworthy. Phosphates are found in excess in the urine in cerebral excitement and meningitis and other diseases involving the nerve centres; in leucæmia, in osteo-malacia and rickets, gout, rheumatism, etc.

Excessive elimination of phosphates takes place in a disease described by Tessier of Lyons as phosphatic diabetes. The symptoms of this disease are not unlike those of diabetes mellitus, there being usually great nervous irritability, rapid emaciation, severe and distressing pains in the lumbar region, more or less dyspepsia, a copious flow of acid urine, but in the place of glucose, phosphates are found in abnormal amounts. There is diminished phosphatic elimination in anæmia, nephritis, diabetes mellitus, cirrhosis, and acute yellow atrophy of the liver, in intestinal indigestion, etc.

Tests.—To detect phosphates put a portion of the urine in a test tube and boil; if a precipitate forms that quickly disappears on the addition of some mineral acid, as nitric acid, the precipitation is due to the presence of phosphates. If a separation of the phosphates is not desired, the test will be made more pronounced by adding a few drops of ammonia to the specimen previous to the boiling of it. A rough idea of the amount of contained phosphates may be obtained by carrying out a similar control test with normal urine.

If it be desirable to separate the phosphates, add to the specimen a solution of ammonia; this will precipitate the earthy phosphates, and they may be got rid of by filtration. If now an ammoniacal solution of magnesia is added to the filtrate, a dense precipitate will form, which will be found to consist of ammonio-magnesic phosphates or the so called triple phosphates.

Ferric Chloride Test.—To several cubic centimetres of urine in a test tube add a few drops of acetic acid, to which add, drop by drop, a solution of 10 parts of water to 1 part of U. S. P. ferric chloride. The presence of phosphates will be proven by the formation of a yellow precipitate. For the quantitative determination of phosphates the reader is referred to special treatises on the subject.

Sulphates.—Free sulphuric acid is not found in the urine. It occurs as the sulphates of sodium and potassium and as the aromatic ethereal sulphates, under which may be included phenol, cresol, pyrocatechin, skatoxyl, and indoxyl. These originate in large part from destructive proteid metabolism in the body and partly from inorganic portions of foodstuff. About 1.50 grammes are eliminated by a healthy individual in the twenty-four hours. The clinical significance of excessive elimination of sulphates is still imperfectly understood. Increased elimination takes place in acute febrile diseases and in those disorders where there is excessive destructive change going on in the tissues. It is found also in pneumonia, acute myelitis, diabetes mellitus, diabetes insipidus, and, in brief, in those conditions which favor increased urea elimination. As would be expected, the sulphates are diminished in the various forms of nephritis.

To determine the presence of sulphates, fill a test tube half full of urine, and add to this one third as much of a solution consisting of barium chloride 4 parts, hydrochloric acid 1 part, and distilled water 16 parts. A dense white precipitate of sulphate of barium will form. By carrying out a similar test with normal urine an approximate estimate of the contained sulphates may be obtained.

Carbonates.—The carbonates of urine are derived from the food, and occur as the carbonates and bicarbonates of sodium, ammonium, calcium, and magnesium. When large quantities of carbonates are found in freshly voided urine, it indicates that the individual is ingesting unusual quantities of carbonates or vegetable acids or is suffering from cystitis. Carbonates may be detected by the addition of any mineral acid. This liberates the carbon dioxide which these salts contain, and in consequence causes effervescence.

Gases.—The chief gases that may be abstracted from normal urine are carbon dioxide and nitrogen. Sulphuretted hydrogen has sometimes been found as an abnormal constituent of urine, but its presence is

usually due to an artificial opening between the intestinal and the urinary tracts. It has also been held to make its appearance through endosmosis without the presence of artificial openings between the intestines and urinary passages. It may be detected by placing a specimen of the suspected urine in a flask and suspending from the stopper above the surface of the fluid a piece of filter paper moistened with acetate of lead; when the gas is present the paper will turn black.

Organic Constituents.—*Urea* (CON_2H_4) is the most important and most abundant nitrogenous constituent of human urine.

Nearly nine tenths of the nitrogen eliminated from the kidneys is contained in the urea that is excreted. The remaining one tenth is eliminated in the form of uric acid, hippuric acid, kreatinin, xanthin, etc. The amount of urea eliminated in twenty-four hours under normal conditions amounts to between 30 and 40 grammes (300 to 600 grains). Urea results from destructive proteid metabolism in the body. It is derived from proteid material contained in the food and from destruction of the proteid-containing tissues of the body. It is formed chiefly in the liver, and in less extent in the spleen and possibly some of the other glandular organs. It is a valuable index of the general nitrogenous metabolism of the body, and in disease the amount of this ingredient thrown off in the urine should be repeatedly determined. It is well to bear in mind that the nitrogen resulting from tissue destruction may, however, under some diseased conditions, be thrown off in some form other than urea. It is also important to remember that in alkaline urine urea is, through ferment action, converted into carbonate of ammonia, hence allowance must be made for this when such urine is found to be deficient in urea. The method of urea formation may, and doubtless does, differ in the several tissues—*e. g.* that formed through the agency of the hepatic cells may come through various intermediate stages from those through which it is formed in the spleen cells, and similarly for the other tissues; but the end product, urea, is the same whether it be elaborated through metabolic processes going on in the hepatic cells, spleen cells, or other cells of the body.

The quantity of urea excreted may be increased physiologically by a rich nitrogenous diet, and conversely food poor in nitrogenous material will cause a diminution of urea. Urea is increased pathologically in fevers, during general inflammatory states, in diabetes mellitus, and in all diseases that increase tissue change. It is, on the other hand, diminished in acute and chronic nephritis, in acute yellow atrophy, cirrhosis and carcinoma of the liver, in malnutrition, and, in brief, in those diseases which impair the general nutrition or destroy the liver cells.

Urea forms no deposit as such in the urine, and hence must be detected either by chemical means or the urine must be treated with some acid—nitric acid, for example—when crystals of urea nitrate will form, and these may be recognized by the aid of the microscope. For convenience, ease of manipulation, and approximate accuracy the Doremus ureameter is an instrument of much value to the physician and may be recommended. It consists of a graduated tube, closed at one end, bent at the other so as to form a neck, and then expanded into a bulb. A standard hypobromite of sodium solution, freshly made according to the following directions, is used:

The hypobromite solution consists of—

Caustic soda c. p., 200 grammes.
Distilled water, 500 c. c.

This fluid is put into the graduated limb of the instrument up to the designated marking. One cubic centimetre of pure bromine is now added by means of a pipette, and enough distilled water is further added to fill the tube to the bend. In this way the hypobromite solution is always freshly made, and this is more reliable. One c.c. of the urine to be tested is drawn up into the graduated pipette that is furnished with the instrument; its tip is passed into the ureameter beyond the bend, and the urine is slowly forced out into the graduated portion of the ureameter. Decomposition of the contained urea quickly takes place, and nitrogen gas gradually collects at the closed end of the graduated limb of the instrument and displaces the liquid. The amount of displacement is then read off on the graduated limb. Each division indicates .001 gramme of 1 c.c. of urine. The percentage of urea present in the urine is found by multiplying the result of the test by 100.

The other more complicated and somewhat more accurate instruments of Hüfner, Dupré, and Liebig may be mentioned, but their method of application is more difficult, and hence they are not practicable for the routine work of the practitioner. If the quantity of chlorides is normal and no sugar or albumin is present, the amount of urea may approximately be estimated from the specific gravity. Thus, 1500 c.c. of urine with a specific gravity of from 1020 to 1024 will contain from 2 to 2.5 per cent. of urea. A specific gravity of 1014, under the qualifications mentioned above, would mean that about 1 per cent. of urea was present, while a specific gravity of 1028 would indicate that 3 per cent. was present. If albumin is present in extent of not more than two tenths per cent., the test is still approximately accurate; if above this, the albumin must be coagulated and removed by filtration before the estimate is made. Urea may be detected qualitatively by evaporating the urine to a syrup-like consistency over a water bath, and adding to a drop of this on a glass slide a drop of nitric acid, when characteristic crystals of nitrate of urea will be found on examination with the microscope. If oxalic acid be used instead, crystals of oxalate of urea will develop.

Uric Acid ($C_5H_4N_4O_3$) forms the next largest and most important nitrogenous constituent of the urine. Like urea, it results from destructive nitrogenous metabolism, and is excreted by the kidneys, appearing as such in the urine. It is formed chiefly in the liver and spleen, and, like urea, is carried in this state by the blood to the kidneys, and is eliminated from the body by the functional activity of these organs. The quantity of uric acid excreted by a healthy adult of average size varies with the character of his diet, but under normal conditions amounts to between 0.5 and 0.75 grammes (7.71 to 11.57 grains).

The quantity eliminated may be increased physiologically by foods rich in nitrogen, and in like manner withdrawal of such foods dimin-

ishes it. The uric acid diathesis, so called, is essentially a disorder of nutrition. The quantity excreted is increased pathologically in fevers, pneumonia, bronchitis, pleurisy with effusion, pericarditis, leucæmia, and in such nervous disorders as chorea, idiopathic epilepsy, *petit mal*, neurasthenia, etc. It is also increased during acute exacerbations of gout, but diminished between the paroxysms. It is lessened in nephritis, diabetes, chronic arthritis, and progressive muscular atrophy. Quinine and antifebrin also diminish its excretion. Since the deposit of uric acid crystals is influenced not only by the degree of condensation of the urine, but by its acidity as well, it is not safe to infer an increased uric acid excretion simply from the amount of uric acid deposit. An accurate estimate can only be made after a careful quantitative study of the entire uric acid excretion for twenty-four hours has been made. For the condition to be abnormal the increase or decrease must be persistent, as is shown by what follows. As desirable as this frequently is, the methods of detection known to us at this time are so intricate and the apparatus so complex that they are not available for clinical work. Herter and Smith and other investigators have developed the fact that there is a normal ratio between the uric acid and urea excreted. This ratio has been found to vary from 1 : 45 to 1 : 65, 1 : 55 being a tolerably fair average. An approach to either 1 : 45 or 1 : 65 indicates that the quantity excreted is either increased or diminished. Ludwig, Salkowski, and others have devised methods for the quantitative estimation of this substance, but for these the reader is referred to special text-books on the subject.

Urates or Salts of Uric Acid.—These consist usually of urates of sodium, ammonium, potassium, calcium, and magnesium. Urates occur in the urine in both the crystalline and amorphous forms.

Urates appear in normal urine after violent muscular exertion and when the urine is condensed, as occurs after excessive sweating. They are also much more apt to make their appearance when the reaction is acid and when the temperature is low. The color of the precipitate may vary in appearance from light gray to pink or brick red. The color will depend in large measure on the degree of concentration of the urine. If the specific gravity is low and the urine pale, the urates will probably be light colored, and *vice versâ*.

The chief clinical significance of an excess of urates is the quantity of uric acid that they represent. About five sixths of the renal concretions found are formed, in large part at least, of uric acid. Urates may appear in the urine in great excess in febrile diseases, rheumatism, gout, hepatic diseases, catarrhal affections of the intestinal tract, in congestion of the kidneys, and after a debauch.

A characteristic of the group is their ready solubility at the bodily temperature; so it is frequently observed that urine which is quite clear when recently passed, quickly becomes cloudy on standing in a cold place. The property which urates possess of dissolving on the application of heat furnishes a valuable test. They may be detected in a given specimen of urine by filling a test tube nearly full and heating the upper stratum. If the cloudiness be due to urates alone, the heated zone will quickly become quite transparent, while the lower one will remain clouded in strong contrast. Occasionally on heating the speci-

men will become clear, owing to solution of the urates, and on heating further to the boiling point a dense white flocculent precipitate will form which does not dissolve on the addition of acid. The last formed precipitate is albumin, which sometimes is found in conjunction with urates.

Urates may also be caused to disappear by the addition of hydrochloric acid, which converts them into uric acid.

Oxalates occur in the urine normally to the extent of 0.3 grains (.02 grammes) in twenty-four hours. They appear sometimes as crystalline formations which do not readily settle to the bottom of the containing vessel, and at other times in solution without the formation of crystals. Oxalic acid is unquestionably excreted by the kidneys, for the crystals have been found in them; it may likewise be formed by decomposition of uric acid and urates after the urine has left the kidneys.

Oxaluria is a condition in which there is persistent abnormal increase of oxalates in the urine, and to determine this with accuracy a quantitative examination of the whole amount passed must be made. For this purpose Neubauer's method is probably the best. Oxaluria is not associated with any particular disease. We know that a diet rich in oxalic acid—*e. g.* tomatoes, asparagus, fresh beans, and beets—will increase the oxalates very greatly in the urine. Oxalates are excreted in excess frequently in dyspepsia. We also know that there is a clinical condition where the excess of oxalates may reach as much as 0.5 gramme (7.71 grains) per litre (.908 quart) of urine, but the only other symptoms of disease, so far as is known, are pains in the back and loins and rapid emaciation. Little is known of the condition from a clinical standpoint, and further investigation along this line is desirable.

Hippuric acid is so called from the abundance in which it occurs in the urine of the horse and other herbivora. Kreatinin (closely allied to kreatin, and resulting from muscular metabolism, being kreatin minus water), xanthin, hypoxanthin, guanin, lactic acid, and phenylic acid occur in the urine of man in very small quantities, but, in so far as is known, their clinical significance is of little moment.

Coloring Matters.—The coloring matter of normal urine is derived from urobilin and urine-indican.

Urobilin—the chief coloring agent—is a dark brown, amorphous substance, resinous in character, and easily soluble in water, alcohol, ether, and chloroform. It probably originates from the action of bile acids on hæmatin, which results from the disintegration of the red blood corpuscles. Its quantity is increased normally after prolonged exertion and excessive sweating, when the urine will be found high colored; and pathologically in fevers, heart and liver diseases, and, in general, in all diseases where there is increased destruction of the red blood corpuscles, as occurs, *e. g.*, in acute febrile states, septic fevers, etc. It is diminished, on the other hand, where the skin activity is lessened by chilling the surface of the body; and pathologically in nervous disorders, hysteria, chronic nephritis, diabetes mellitus, intracranial hemorrhage, hæmatocele, extra-uterine pregnancy, and generally in those diseases where there are diminished production and destruction of red blood corpuscles, as occurs in anæmia, chlorosis, and convalescence from acute and exhausting diseases.

Urine-indican is obtained by the absorption of indol from the intestines. Indol results from imperfect pancreatic digestion; so when this digestive disorder exists, indol is formed in excess, a greater amount of it is absorbed, and more than the usual amount of indican appears in the urine. *Indicanuria*, or the appearance of indican in excess in the urine, gives evidence of albuminous putrefaction or excessive fermentation in the alimentary tract. It may also occur when proteid fermentation is going on in other parts of the system. Putrefactive bacteria play an important rôle in the production of this disorder. It is of frequent occurrence in diarrhœas and some forms of constipation—in tuberculosis, pyothorax, carcinoma of the liver, Addison's disease, diabetes mellitus, and other disorders where albuminous putrefaction is going on. It may be detected by adding drop by drop to a mixture of equal parts of urine and hydrochloric acid a saturated solution of calcium hypochlorite. A blue reaction develops, due to the formation of indigo, and this may be separated by shaking the mixture with chloroform. Too much hypochlorite must not be added, as it will decolorize the indigo. The chloroform will dissolve the indigo and then hold it in solution as a blue zone upon the stratum of liquid below.

Choluria.—Bile Acids.—Traces of bile acids are to be found in normal urine. Bile acids occur in the urine of jaundiced patients, in the urine of individuals having tumors of the liver, cirrhosis of the liver, in acute yellow atrophy of the liver, in phosphorus-poisoning, etc. The tests for isolating bile acids and proving their presence are so complicated and tedious that they are scarcely available for clinical use. Pettenkofer's test consists in adding concentrated sulphuric acid to the specimen of urine and keeping it at a temperature not higher than 60° to 70° C. To this a 10 per cent. solution of cane sugar is added drop by drop, and the mixture is constantly stirred. The liquid will assume a beautiful red color if bile acids are present. Even this test is not always reliable, and if the urine be deeply colored it cannot be applied without going through processes too long and tedious to be practically available.

Bile pigments are frequently found in the urine of jaundiced patients. They may come directly from morbid changes in the blood causing rapid and extensive disintegration of the red blood corpuscles, as occurs after hemorrhage into the tissues, or they may arise from the bile which accumulates in the liver from obstruction of the bile ducts; the bile is taken up by the lymphatics, gains entrance to the blood stream, and is removed from the blood by the kidneys. Urine containing bile pigments varies in color from yellow to dark green. It is usually fairly clear, and on shaking beads of yellow froth accumulate on the surface. Bile pigments may occur in the urine before pigmentation of the skin is apparent; hence their detection is important. Of the many tests for their identification, three may be mentioned which are valuable because of their ease of application and accuracy:

1. *Gmelin's Test.*—1 c.c. of pure fuming nitrous acid is put into the bottom of a test tube, and 1 c.c. of urine is, by means of a pipette, made to trickle down the side of the test tube, so that the latter will float upon the surface of the former without mixing with it. At the juncture of the two fluids a beautiful play of colors of the spectrum

will be observed if bile is present, the colors varying from yellow to blue, violet, red, and green. The green is essential to prove the presence of bile, but the others, in atypical cases, may be obscured or absent. The test will not be available if the urine has been treated previously with alcohol, since it produces a bluish green tinge when brought in contact with nitrous acid.

2. A *modification* of this test consists in filtering a quantity of urine through a filter paper that is free from impurities, and placing a drop of nitrous acid on the filter paper. If bile is present, the same play of colors will develop about the drop of acid.

3. Again, a drop of urine and a drop of nitrous acid may be placed near each other on a white porcelain plate. If bile is present, the same exhibition of colors of the spectrum will take place when the drops of urine and acid are joined together by a clean glass rod.

Mucus.—In the passage of the urine from the kidneys to the bladder, and thence out through the urethra, a certain amount of mucus is normally added to it by the cells located in the mucous membranes over which it passes; so that normal urine on standing for a time will develop a slight cloud or smokiness due to the contained mucus. When this substance is greatly increased in quantity, it is due to catarrhal or purulent inflammation of one or more of the mucous surfaces over which the urine passes. In cases of severe cystitis the mucus may be so abundant and of such tenacious consistence that it adheres tightly as a sheet to the bottom of the vessel, so that it has to be removed with a stick or mop. When mucus is present in large amount pus corpuscles also occur in considerable quantity, and the urine is invariably alkaline. To free a given specimen from mucus precipitate the mucus with acetic acid and filter the urine. The clear urine will then pass through the filter and the mucus will remain behind. It is necessary frequently to do this, because the mucus will at times obscure the reactions for albumin.

Blood is always an abnormal ingredient of the urine, and when present may give to it a color varying from light brown to a smoky, dirty red or even bright scarlet. Its color cannot in some instances afford positive proof that the urine does contain blood; hence chemical and microscopical tests are essential to establish the fact. When found in the urine it indicates disease or injury to some part of the urinary tract. It may come from acute inflammatory disease of the kidneys, ureters, bladder, or urethra; from wounding some of these by external violence—*e. g.* by the passage of a renal calculus; the presence of irritating parasites, drugs, malignant growths; or by ulcerated processes occurring in any portion of the urinary tract. When the amount of blood is excessive it is easy of detection. It may be present in sufficient quantity to collect in large clots in the bladder, and these may be passed in considerable size when the urine is voided. On boiling a specimen of urine in which blood is present to any marked degree it will coagulate, because of the contained albumin. For the detection of blood chemically Almen's test may be recommended. To carry this out take 10 c.c. of urine in a test tube and pour on its surface gently, without mixing, a mixture of equal parts of tincture of guaiacum and oil of turpentine. If blood is present, there will occur at the line of contact

of the two liquids a white ring which gradually turns blue. It may also be detected by the spectroscope and by the microscope. For other tests the reader is referred to special works on this subject.

If on examination by the microscope blood corpuscles are found, the diagnosis of hæmaturia is established.

Hæmaturia is the passage of blood plasma with the corpuscles into the urine. It is diagnosticated by the large amount of sediment, by the chemical tests given above, and by the microscope, which reveals the corpuscles either unaltered, or their disks with sometimes an alteration in shape, or with the coloring matter dissolved out. It occurs in any disease which causes active congestion or destroys the integrity of any portion of the mucous membrane of the urinary tract—*e. g.* in scarlet fever, acute nephritis, and in certain acute fevers as, yellow fever. It may also occur in acute catarrhal and ulcerative conditions of the urinary mucous membrane. When the blood comes from the kidneys the color is darker, it is evenly diffused throughout the urine, and casts and renal epithelium are usually present.

Hæmoglobinuria occurs when the hæmoglobin is dissolved out of the red blood corpuscles and appears in the urine. In this condition the stroma of the corpuscles is either absent or present in too small amount to account for the hæmoglobin found. It occurs in the course of certain acute infectious diseases—*e. g.* erysipelas; in malarial fevers, especially in hemorrhagic malarial fever, where the absence of the corpuscular disk makes a valuable differential diagnostic sign in distinguishing between it and yellow fever; in rheumatism and sometimes syphilis; after extensive burns and attacks of cold, and after the introduction of various poisons into the system—*e. g.* naphthol, potassium, chlorate, etc.

Albumin.—Luebe and Winternitz have proved that the urine of normal individuals is free from albumin, and hence when it is found in the urine in even small quantities in the apparently healthy it is fair to assume that it is in a sense abnormal, and is a transient perversion of the normal physiological function due to circulatory disturbances. It has been shown experimentally that albumin does not readily dialyze through animal membranes. For albumin to appear in the urine there must be change in the composition of the blood, increase of blood pressure in the kidneys, or structural degeneration of these organs. One or all of these causes may be present in the same case. When the albuminuria is brought about by destructive changes in the kidneys themselves, it is always a source of serious concern.

It is therefore well to bear in mind that the mere presence of albumin in the urine does not necessarily imply *renal disease*. Extra-renal albuminuria may be caused by injury or disease to any portion of the urinary tract from the renal pelvis to the urethra. If the urine contains albumin, but no casts or renal epithelium, and many pus cells, it is probable that the albumin comes from some portion of the urinary tract outside of the kidneys. The quantity of albumin found in a given specimen of urine is not always an index of the severity of the renal lesion, since only small quantities may be found in red atrophy and cirrhosis of the kidneys. Albumin appears in several forms in the urine. It may appear as serum-albumin, globulin, peptone albumose, hæmoglobin, fibrin, or mucin. These may all be separated by appropriate

methods, and are of much value from a diagnostic standpoint. Albumin may be caused to appear temporarily in the urine by diet, by excessive exercise, by drugs, and by certain diseased conditions.

Albuminuria may be produced by circulatory disturbances, and hence is found in various forms of heart disease and in certain diseased states of the arteries—*e. g.* in general arterio-fibrosis. It may also be produced by many if not all of the acute febrile diseases. It is almost uniformly present in pneumonia, scarlatina, and diphtheria. It occurs in diseases affecting the composition of the blood, as, *e. g.*, in scurvy, purpura, leucæmia, and anæmia. Albuminuria may be produced also by poisoning with such drugs as cantharides, carbolic acid, phosphorus, and turpentine. Finally, it is generally found in acute and chronic renal disease. When the albuminuria is due to destructive changes in the kidneys, the microscope will almost invariably show tube casts and renal epithelium. Serum-albumin and globulin, while capable of separation, when occurring in the urine are usually found together, and for practical purposes need not be dissociated. The methods for albumin detection are many, but only the four most reliable and available ones will be discussed here.

In all cases where the urine is cloudy, it should be filtered.

1. *Heat and Nitric Acid Tests.*—For the performance of this, a test tube should be filled to within one inch of the top, and the uppermost portion should be boiled in the flame of a spirit lamp or Bunsen burner. When urine is thus boiled one can compare the heated zone with that below it, and the method is hence more accurate. On applying heat to a given specimen of urine, if it be normal no visible effect beyond the ebullition due to the boiling will be observed. If the urine is turbid from the presence of suspended urates, the application of heat will quickly cause these to be dissolved, and the heated zone will become quite transparent. If the urine is alkaline, it must be made faintly acid by the addition of a few drops of acetic acid. This will cause precipitation of the mucus, which may be removed by filtration. Too much acid may convert the contained albumin into soluble acid albumin, not coagulable by heat, in which case the test will fail, and the same is true on the addition of nitric acid. If now, on applying heat to the upper stratum of urine in a test tube, a cloudiness appears, this cloudiness may be due to one of two things—viz. phosphates or albumin.

If the cloudiness be due to phosphates, the addition of nitric acid, drop by drop, will cause them to quickly disappear and the urine to become transparent: if due to albumin, the acid will not only fail to dissolve the flocculent white precipitate, but may intensify it.

2. *Heller's Test.*—Place 1 c.c. of reduced nitric acid in a test tube, and upon its surface float, without mixing, 1 c.c. of urine. If albumin is present, a white ring like dense smoke will form at the junction of the two liquids. The size and density of the ring will depend on the quantity of albumin present. The cloud may not appear at once if the amount of albumin is small, so that the tube should be set aside for twenty or thirty minutes, at the end of which time a faint though sharply defined whitish ring will form if albumin be present. This test is subject to two errors that may be avoided by proper precautions—viz. amorphous urates will form a smoke-like ring at the point of con-

tact of the two liquids, but the zone when formed by urates will disappear upon the application of heat; and, again, mucin will make a ring similar to that formed by albumin, but it has already been pointed out that this substance may be precipitated by acetic acid, and gotten rid of by filtration before this test is applied.

3. *The Ferrocyanide Test.*—This test is commended by Purdy and others for its accuracy and simplicity. The test is carried out as follows: To a test tube half filled with urine add 1 to 2 drachms of a 1 : 20 potassium ferrocyanide solution. Thoroughly mix these and add 10 to 15 drops of acetic acid, when a cloudiness or dense flaky precipitate will form. The density of the precipitate will be regulated by the amount of albumin which the urine contains.

FIG. 55.

Esbach's albuminometer.

4. *Picric Acid Test.*—This is conveniently performed by means of Esbach's albuminometer. The process is conducted as follows: Pour the urine to be examined into the thick graduated test tube up to the point *U*, then pour in up to the point *R* a solution made according to the following formula: 10 grammes of picric acid, 20 grammes of citric acid, and enough boiling water to make 1000 c.c. Thoroughly mix the urine and reagents and set the tube aside for twenty-four hours. The contained albumin will settle to the bottom as a thick, yellowish white precipitate, and the quantity may be read off on the graduated scale, the figures indicating the grammes of dried albumin in 1000 c.c. of urine. This process is subject to error when the urine contains quinine, antipyrin, and thalline.

Globulinuria is a condition which is never present alone, but occurs in combination with serum-albuminuria, so that what has just been said concerning the presence of serum-albumin in the urine will apply to this condition.

Peptonuria.—When the kidneys eliminate peptones and these appear in the urine, we have the condition known as peptonuria. It has also been called pyogenic peptonuria, from the fact that the condition is brought about by diseases which cause accumulation and destruction of the white blood corpuscles, in which diseases peptones and other disintegration products are liberated. Consequently, as would be expected, peptonuria occurs in the resolving stage of pneumonia, in pyo-thorax, pyo-pneumo-thorax, in purulent meningitis, acute articular rheumatism, in tuberculosis, syphilis, scurvy, otitis media, and, in general, in conditions where extensive suppurative changes are going on, and where the conditions are favorable to absorption of their products by the blood. It has also been shown that in ulceration of the intestines the peptones derived from the action of the digestive juices on proteids may pass directly into the blood through the ulcerated part or parts, and cause the appearance of peptones in the urine. Peptones, according to Fischel, appear normally in the urine in the puerperal state. The appearance, then, of peptone in the urine would lead one to infer that suppurative pro-

cesses are going on somewhere in the body, and its detection as a diagnostic point is of no small value. It is necessary in making a diagnosis to be able to exclude pregnancy and ulceration of the intestinal tract.

The tests of Hoffmeister and Devoto, while accurate, are too tedious for clinical work. If present in large amount, peptone may be detected by the biuret reaction, or a better and more delicate test consists of the following procedure: Make the urine slightly acid with acetic acid, and add ammonium sulphate to saturation, filtering off any precipitate that may form. The addition of picric acid or potassio-mercuric iodide will cause precipitation of peptones if they be present. This test hangs on the fact that peptones are not precipitated by nitric acid or by saturation with ammonium sulphate, as other albumins are.

Fibrinuria may be present when there is inflammation with exudation in any portion of the urinary tract. The fibrinous exudate is then washed off and appears in the urine.

Chyluria is a condition in which fat, albumin, pus cells, and not infrequently red blood corpuscles make their appearance in the urine. Casts are not present unless the disease excites nephritis. The quantity of these abnormal elements will depend upon the severity of the disease underlying the condition. Chylous urine has a peculiarly characteristic appearance which is no small clue to the diagnosis. It has a distinctly milky look, which may be tinged pink from the presence of red blood corpuscles. Such urine tends to coagulate after being voided, and the condition may be so severe that complete gelatinization occurs throughout, the coagulum taking the shape of the containing vessel. Coagulation may even take place within the bladder, when it causes great distress because of the inability to pass the clot until it is broken up. It was formerly believed to be a disease peculiar to the tropics, but cases have occurred in individuals who have never resided in the tropics. Investigation has developed the fact that in the tropics, at least, the disease is produced by the entrance of filaria sanguinis hominis or its ova into the system by means of the drinking water. The body then becomes the host of the parasite, which makes abnormal connecting passages between the lymphatic system and the urinary tract; hence the appearance of chyle in the urine. On heating such urine it coagulates from the presence of the contained albumins, and when shaken up with ether the fat will be dissolved and the fluid may become quite clear. By the aid of the microscope the fat and other elements, including the filaria sanguinis hominis and its ova, may be discovered.

Since abnormal connecting passages may be produced by this parasite between the lymphatics and the urinary tract, it does not seem unreasonable to assume that ulcerative and necrotic processes may form similar channels of communication, thus producing a chyluria entirely similar to the former except for the absence of the parasite.

Glycosuria.—Glucose ($C_6H_{12}O_6H_2O$) is sometimes present in minute quantities in the urine of persons in apparent health. Even this small trace is absent from the urine of some, and it may well be doubted that such elimination is strictly normal. It is more probable that there is a perversion of the normal physiological function that does not give rise to any morbid symptoms beyond the appearance of this abnormal ingredient in the urine in minute quantities. It may also appear tran-

siently in greater amount from the ingestion of large quantities of carbohydrate foods and from other temporary derangements. It must be present in considerable quantity and habitually to constitute the diseased condition known as diabetes mellitus. Glycosuric urine is usually pale in color, of acid reaction, high specific gravity, sweetish taste, somewhat syrupy consistence, and favors the rapid development of the yeast fungus or *torula cerevisiæ*.

While it is true that urine which contains much sugar is of high specific gravity, and most authorities advise that when there is a specific gravity of 1028 or over it should be tested for sugar, it is nevertheless also true that glucose may occur in urine of normal or even subnormal specific gravity, so that to be accurate all urines should be tested for this substance in order to eliminate it beyond question. Of the many tests for sugar, only a few of the most reliable ones will be mentioned here.[1] It is well to bear in mind in testing for this ingredient that albumin obscures the test, and hence it must be got rid of by coagulation and filtration before the tests for sugar can be applied. Among the tests for sugar in the urine may be given—

A. Fehling's test, which is composed of the following ingredients: (1) *The copper solution*, consisting of 34.64 grammes crystallized c. p. copper sulphate dissolved in enough distilled water to make 500 c.c. (2) *The alkali solution*, consisting of 173 grammes c. p. recrystallized Rochelle salt and 60 grammes c. p. caustic soda, dissolved in enough distilled water to make 500 c.c. To apply the test take 1 c.c. of the No. 2 solution in a test tube, and pour into it 1 c.c. of the No. 1 solution, and boil; to this while boiling add the suspected urine drop by drop. If grape-sugar be present, the copper sulphate will be reduced to cuprous oxide, and a bright opaque yellow or orange precipitate of the suboxide of copper will quickly form. This will, on standing, settle to the bottom as a reddish precipitate. If very little sugar is present, it may be necessary to add quite a number of drops of the suspected urine, but the amount should not be greater than that of the Fehling solution. In employing this test a portion of the mixed urine for twenty-four hours should be taken, and in gaining any idea of the quantity of sugar excreted the copper sulphate must be entirely reduced. Furthermore, the urine should not be allowed to stand long before it is tested, else fermentation may set in and break up much of the contained sugar into carbon dioxide and alcohol. The test is especially useful to the physician, because by watching the point at which all the copper is reduced, and remembering that 200 grains of the solution are decomposed by 1 grain of sugar, the quantity of sugar excreted may be estimated with approximate accuracy.

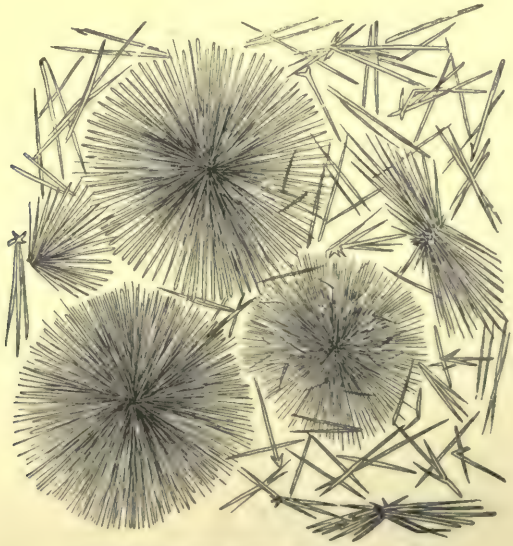
To determine the quantity of sugar present take 1 cubic centimetre of the Fehling solution and dilute it with 4 parts of distilled water. Boil this in a beaker, and if no precipitate is formed the purity of the solution may be assumed. Now, to the boiling Fehling solution thus diluted add $\frac{1}{10}$ of a cubic centimetre of urine from a graduated burette. Continue to add the urine in this amount until all the blue color shall have left the Fehling solution, and in its place the yellow color charac-

[1] I have found sugar in considerable quantity in urine with a specific gravity as low as 1010.

teristic of the suboxide of copper shall have formed. If 1 c.c. of urine shall have been required to attain this result, the urine contains $\frac{1}{2}$ of 1 per cent. of sugar. If 2 c.c. of urine are required, it will contain $\frac{1}{4}$ of 1 per cent., and so on. This test is subject to some error, but is fairly accurate.

2. *Roberts's fermentation test* is also most excellent, and its ease of application makes it especially useful to the practitioner. It depends on the action of the torulæ in the saccharine urine, breaking up the sugar into carbon dioxide, which is given off as gas, and leaving behind alcohol, which has a specific gravity less than water. Urine, therefore, in which this fermentation has taken place is of lessened density, and Roberts has discovered that every degree of density lost represents 1 grain of glucose to the ounce of urine. To apply the test: Take 4 ounces of the mixed urine for twenty-four hours, and put into a 12-ounce bottle with a block of Fleischman's yeast. The bottle is then imperfectly corked, so as to allow the carbonic acid gas to escape, and is set aside in a warm place for twenty-four hours, in which time the fermentation will be complete. As a control experiment a 4-ounce vial filled with urine without any yeast and tightly corked is set beside the first. At the end of twenty-four hours the specific gravity of the two specimens is taken. If the specific gravity of the fermented urine is 1020, while that of the unfermented is 1030, then 10 grains of sugar to the ounce of urine are present, and by multiplying the number of grains lost by .23 the percentage may be approximately obtained.

FIG. 56.



Crystals of phenyl-glycosazon (Von Jaksch).

3. *Phenyl-hydrazin Test.*—This is perhaps the most delicate test yet discovered for the qualitative determination of sugar in the urine. Phenyl hydrazin in the presence of sugar forms characteristic yellow

needle-like crystals which frequently arrange themselves in the form of a rosette. To apply the test, take 2 parts of hydrochlorate of phenyl hydrazin and 3 parts of acetate of soda in a test tube, and add 6 to 8 c.c. of urine. If warming does not dissolve the salts, add a little water and place the tube in boiling water for twenty or thirty minutes. After this put the tube in a cold place. If simply a trace of sugar is present, a yellow crystalline deposit of phenyl-glucosazon will develop. These may easily be recognized by a lens magnifying not more than 200 diameters.

Acetone (C_3H_6O) is found in mere trace in the urine of healthy individuals. It is a colorless liquid possessing a peculiar fruit-like odor and a specific gravity of 0.792. It may be increased by excess of nitrogenous food. When present alone it need cause no alarm, but occurring as it does with diabetes mellitus it indicates an advanced stage of the disease, and is of very serious import. When accompanied or succeeded by diaceturia the prognosis is exceedingly grave.

Acetonuria is the appearance of acetone in excess in the urine. It may occur in all fevers where there is much blood change, and the quantity of acetone excreted will generally correspond with the degree of temperature elevation. It frequently occurs in the urine of patients suffering with carcinoma; in inanition, in severe cerebral excitement, in derangements of the digestion, and from auto-intoxication. It in like manner occurs in the urine of patients suffering with smallpox, typhus fever, pneumonia, scarlet fever, measles, nephritis, etc.

Tests.—Lieben's iodoform test may be recommended as easy of application and reliable for even small traces of acetone. To apply it, proceed as follows: To several c.c. of urine add a few drops of iodo-potassic iodide solution and caustic potash. If acetone be present, an abundance of iodoform crystals will quickly develop.

Legal's nitro-prusside and caustic potash test may also be mentioned as being fairly accurate and easy of application.

Diaceturia is the appearance of ethyl diacetic acid ($C_6H_{16}O_3$) in the urine. It is always an abnormal condition, but when occurring, as it frequently does, in the urine of children suffering with fever is not of serious clinical import. It frequently occurs in connection with acetone in fevers and in diabetes in adults. V. Jaksch has observed it to precede coma in these cases several days, and the resulting coma is, in his opinion, due to the diacetic acid. Diacetic acid may also be found in the urine of patients suffering with pericarditis, pleuritis, perityphlitis, typhoid fever, miliary tuberculosis, pulmonary phthisis, and pneumonia.

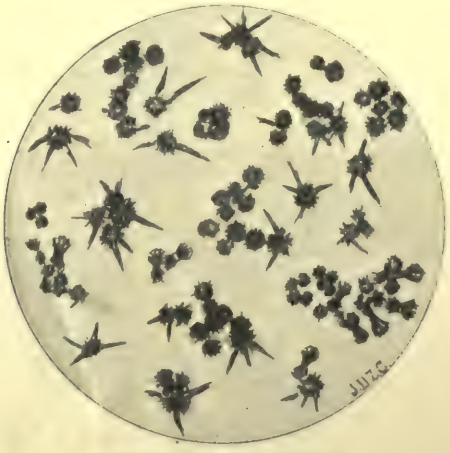
Test.—To several cubic centimetres of urine add a concentrated solution of ferric chloride drop by drop, when any phosphatic precipitate that forms must be removed by filtration and more of the ferric chloride added. If now a Bordeaux red color appears, boil one portion of the urine, and to another add a few drops of sulphuric acid and shake with ether. If the boiled urine does not change perceptibly, and if after twenty-four to forty-eight hours no ferric chloride reaction is found in the ethereal extract, the condition of diaceturia is proven (Von Jaksch).

Microscopical Examination of the Urine.

Method of Detection.—In every microscopical examination of the urine, just as in the chemical analysis of it, a portion of the entire amount for twenty-four hours should be taken, for just as the substances detected by chemical means may be excreted in diminished amounts or withheld at certain periods of the day, so will the microscopical ingredients vary, appearing at some times and being absent at others. To this end the urine should be put into a tall conical glass vessel, and covered to prevent the entrance of extraneous particles from the air. By setting this aside for ten or twelve hours the suspended particles will have opportunity to settle to the bottom, from which they may be taken by means of a pipette and examined. As the urine undergoes change on standing, a quicker and more accurate method is secured by the use of a centrifugal machine, the sedimentator. The instrument consists of metal tubes hanging loosely by pivots in a horizontal plate. In one of these metal holders a glass tube containing the urine is placed and the plate is made to revolve rapidly. The rapid revolution causes the holders to assume a horizontal position, and centrifugal force causes the contained solids to settle quickly to the bottom of the glass tube. The sediment may now be withdrawn by means of a pipette and examined.

The most abundant and perhaps the least important sediment found in the urine is composed of urates. As already stated, these tend to form in urine that is concentrated, of acid reaction, and where the temperature is low. Urates occur in certain febrile, renal, and hepatic disorders, and the quantity varies within wide limits. The color of the deposit will depend upon the degree of concentration and color of the urine in which it occurs. In pale urine with low specific gravity it may

Fig. 57.



Ammonium urate (Musser).

be quite light, while in urine that is deeply colored and of high specific gravity it may be pink or a brick red.

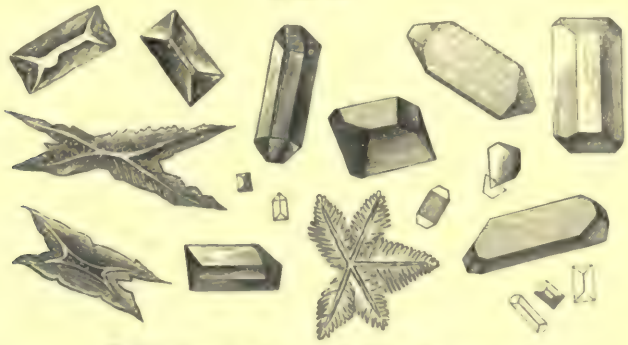
Amorphous urates occur as a granular deposit which may be dis-

tinguished from trash and other detritus by their rapid disappearance on heating and on the addition of acids.

Urates of Sodium and Ammonium.—Urate of sodium occurs in urine that is acid or just changing to alkaline as a crystalline deposit of yellowish spheres, with spicules very frequently attached to them, and occasionally as dumb-bell crystals. Their color ranges from light to yellowish red. When occurring in alkaline urine, as they sometimes do, in the shape of the so-called thorn-apple crystals, they may be regarded as crystalline forms of urate of ammonium. Acid urates of potassium and calcium may also occasionally be formed.

Tribasic Phosphates.—Triple phosphates, ammonio-magnesium phosphates ($MgNH_4PO_46H_2O$), precipitate to some extent occasionally in feebly acid urine, more commonly and in greater abundance in alka-

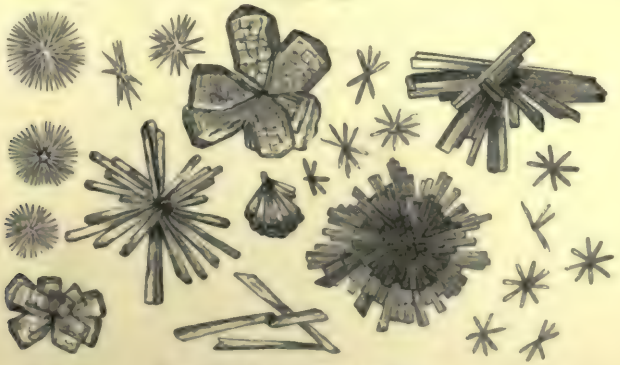
FIG. 58.



Various forms of triple phosphates (Finlayson).

line urine. They frequently occur as a dense white crystalline deposit. The crystals under the microscope appear as transparent prisms with bevelled edges, as the characteristic knife-rest or coffin-lid crystals. There are many modifications of this form, some being arranged like a

FIG. 59.



Crystalline phosphates (Finlayson).

star, the spangles of which are made of feather-like crystalline formations.

Phosphate of calcium (Ca_3Po_4) occurs in feebly acid and alkaline

urine. It appears as an amorphous deposit or as conical or wedge-shaped crystals, which occur singly or in groups, making a rosette shape; the apices of the cones point to the centre. They occasionally occur as spherules or in dumb-bell shapes. The latter crystals may be distinguished from the similar ones of uric acid by the fact that they

FIG. 60.

Crystal forms of calcium oxalate; × 275 (Eichhorst). In the centre, common octohedra; at the margins, discoidal hour-glass and dumb-bell forms.

quickly disappear on the addition of acetic acid, while the uric acid dumb-bells dissolve in alkalies.

Basic phosphate of magnesia may occur as strongly refracting, somewhat elongated rhombic plates in feebly acid neutral or alkaline urine.

FIG. 61.

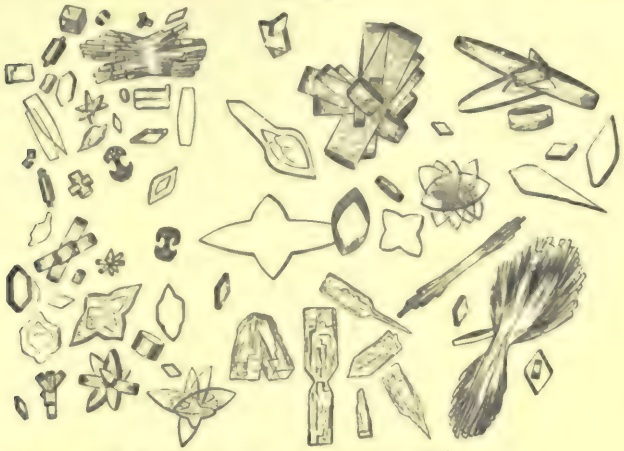
Calcium sulphate crystals (Musser).

Oxalate of calcium ($CaC_2O_2 2H_2O$) occurs in acid and rarely in alkaline urine in two crystalline forms—viz. the characteristic octahedral or envelope crystals and as dumb-bell crystals. They do not readily settle

to the bottom of the fluid as a sediment, but are more apt to remain suspended in it. They may frequently appear in normal urine, and are increased by food rich in oxalic acid, such as tomatoes, asparagus, beets, fresh beans, etc.

Carbonate of calcium occurs in alkaline urine as an amorphous

FIG. 62.

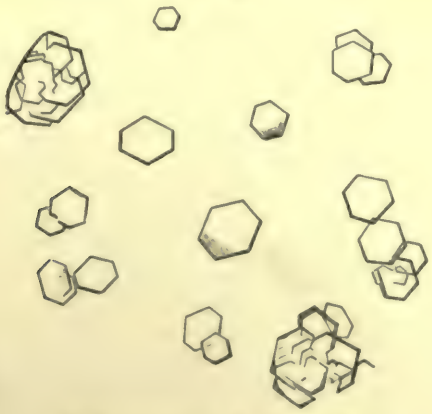


Various forms of uric-acid crystals (Finlayson).

powder which is quickly decomposed by mineral acids, giving up carbon dioxide.

Sulphate of calcium occurs rarely in human urine. It forms in

FIG. 63.



Cystin crystals; × 275 (Eichhorst).

acid urine with high specific gravity in needle-like prisms, and has no clinical significance.

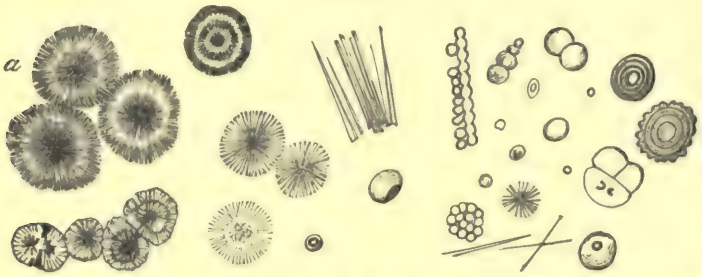
Uric acid crystals appear in acid urine. They crystallize in various shapes, the characteristic whetstone and rhombic tables with rounded edges being commonest. They may be recognized by their deep brown-

ish yellow color and by the fact that they quickly dissolve in caustic potash.

Hippuric acid crystals may appear in acid urine after the ingestion of cranberries and other fruits and after the administration of benzoic acid. They appear as long four-sided prisms that are soluble in ammonia.

Cystin ($C_3H_7NSo_2$) crystals are rare in human urine. They occur in acid urine as six-sided plates which frequently overlap each other.

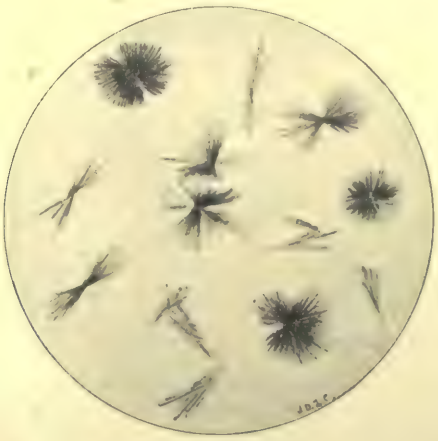
FIG. 64.



Crystals of leucin (different forms) (crystals of creatinin chloride of zinc resemble the leucin crystals depicted at *a*.) The crystals figured toward the right consist of comparatively impure leucin (from Charles: *Physiological and Pathological Chemistry*).

Leucin and **tyrosin** crystals are usually found together. They may be present in urine that is loaded with bile pigment. Leucin crystals are yellow spheres that are marked with concentric radiating lines.

FIG. 65.



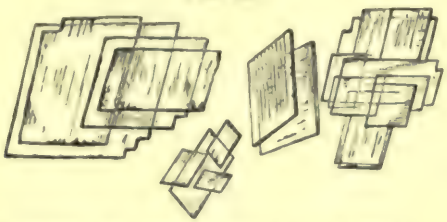
Tyrosin crystals (Musser).

Tyrosin occurs as very fine needle-like crystals gathered together like a bunch of wheat straw.

Indigo.—Sometimes needles and feathery crystalline forms of indigo may be found in the urine of jaundiced patients and in cases of cirrhosis and abscess of the liver. The blue color will usually be sufficient to distinguish these crystals.

Cholesterin crystals are flat four-sided scales. They usually appear in masses superimposed one upon the other. According to Pöhl, they are found in the urine of patients having fatty degeneration of the kidneys, jaundice, chyluria, and diabetes. They are rare formations in the urine, and Von Jaksch has found them but once, and then in a patient suffering with tabes and cystitis.

Fig. 66.



Crystals of cholesterin (from Charles: *ibid.*).

Organized Sediment.—Under this classification may be included pus, blood, epithelium, renal tube casts, spermatozoa, micro-organisms, elements of morbid growths, and parasites.

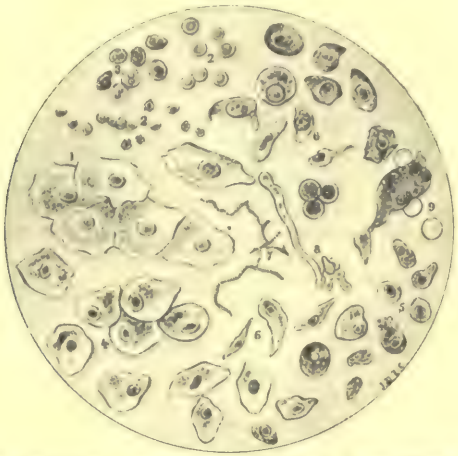
Pus cells may frequently be found in small numbers in normal urine. These do not differ in any essential detail from those found in other parts of the body. It is only when they occur in large numbers, and usually with much mucus, that they may be considered pathological. They may be derived from any portion of the urinary tract from the renal cortex to the end of the urethra. They are found in greatest abundance in catarrh of the bladder, in pyelitis, and in acute catarrhal urethritis.

Red blood corpuscles appearing in the urine may be quite normal in appearance, or the hæmoglobin may be partially or entirely dissolved out, or they may be crenated. The appearance of the corpuscles may give some idea of their place of origin. When normal or slightly changed they come, as a rule, from the urethra or bladder; when intimately mixed with the urine and settling slowly to the bottom of the vessel appearing as pale or colorless disks (the phantom corpuscles, so called), they are probably derived from some portion of the urinary tract higher than the bladder—*i. e.* from the ureters or kidneys. They are present in the urine in acute general febrile disturbances—*e. g.* in scarlet fever, yellow fever, in acute nephritis, and in the exacerbations of chronic nephritis, and, in general, in all acute inflammatory conditions of the mucous membrane of the urinary tract. Morbid growths, injuries, and destructive changes in the urinary mucous membrane may also cause their appearance in the urine.

Epithelium.—A few desquamated epithelial cells may be expected to be found in normal urine. Their presence in great numbers constitutes a pathological condition. There may be large flat squamous epithelial cells from the bladder or urethra, or smaller ones derived from the ureters and pelvis of the kidneys. The cells present a great variety of size and structure—so much so that their place of origin cannot always be positively identified. When present in great numbers they indicate

cystitis, but beyond this one cannot determine these pathological conditions with accuracy. Their number and appearance, however, are valuable in connection with other clinical signs. There are sometimes present certain cuboidal cells (somewhat smaller than those found lower down

FIG. 67.



Cellular elements from the urine: 1, squamous epithelium; 2, red blood corpuscles; 3, polynuclear leucocytes; 4, transitional cells; 5, epithelium from the kidneys; 6, epithelium from the bladder; 7, micrococcus aureæ; 8, yeast fungi.

in the urinary tract) having single prominent nuclei and occurring singly or in cylindrical arrangement in the form of the tube from which they were exfoliated. Such cells come from the convoluted tubules, and when appearing singly they are aids to diagnosis, but, like the others, cannot be absolutely depended upon. When superimposed upon the surface of the tube cast they are certain evidence of renal disease.

Renal tube casts when present in the urine are of the greatest clinical importance. Authorities differ on the point, but with our present knowledge it is fair to assert that tube casts never appear in perfectly healthy urine. That certain varieties do occur transiently and in conditions of apparent health cannot be denied, but the state must be one of apparent normality only, for there must be perverted physiological function, even though slight and temporary, for such variation from the normal to occur.

Renal tube casts may be divided into blood, pus, epithelial, granular, fatty, hyaline, and waxy casts. If one considers the mode of formation of tube casts, it is apparent that blood, pus, epithelium, and granular débris may all be found in a single cast, in which event the preponderating element will serve to place the casts under one of the above subdivisions. It is more than probable that no true casts are thrown off without some inflammatory change having taken place in or around the uriniferous tubules. Whatever the nature of the cast, after a time the mass shrinks somewhat, and is then easily washed down into the bladder by the urine. When blood casts are formed there is previous abnormal dilatation of the capillaries surrounding the uriniferous tubules, and in

the glomeruli the corpuscles, together with the coagulable elements of the blood, escape from the capillaries, collect in the tubules, there to be moulded into casts, after which shrinking takes place, and they are washed out by the urine into the renal pelvis and thence into the bladder. Pus casts may originate in a manner not dissimilar, probably when there is a lower grade of congestion. On the other hand, the congestion may be so great as to interfere with the nutrition of the cells lining the kidney tubules, causing them to be exfoliated and form epithelial casts, or these cells and the extravasated blood corpuscles may disintegrate, resulting in the formation of the granular variety. Not infrequently the cells composing the epithelial casts will show evidence of fatty degeneration, thus giving a clue to the kind of change going on in the kidneys. The number of tube casts in a given specimen of urine will depend on the activity of the pathological change taking place in the kidneys, there being an increase in number in acute nephritis and in the exacerbations of the chronic form, while the number of casts thrown off in the intervals between the attacks will be greatly diminished. If care be exercised to secure a complete settling of the solid elements of the urine before making an examination, the number of casts found will be a valuable index to the stage of the disease. Tube casts are not generally more than $\frac{1}{25}$ inch long, and their width will vary with that portion of the tubules from which they come.

Blood casts are more or less granular, from the fact that some disintegration of the epithelial and corpuscular elements takes place in the tubule, but, as the name indicates, the red blood corpuscles make the striking feature of the casts, and these with the granular detritus are held together by the fibrin filaments that develop in the tubule. They are easily identified with the microscope, and occur when there is great renal congestion or capillary hemorrhage produced by any disease or lesion.

Pus casts are also somewhat granular, cylinders having the same fibrinous exudate for a basis as the blood casts above described, but instead of red blood corpuscles they contain extravasated leucocytes in great excess. Their mode of formation and cause need not differ essentially from blood casts, and it not rarely happens that both red and white blood corpuscles are found entangled in the same specimen.

Epithelial casts occur as aggregations of the exfoliated cells of the tubules of the kidneys. There is usually formed first a fibrinous mould in the tubule, and upon this the epithelial cells adhere. Any diseased condition that would contribute to interfere with the proper nutrition of the epithelial lining of the tubules would be an efficient cause in the production of this variety of casts. The exfoliation of the cells may take place singly or they may be thrown off *en masse*. These casts usually occur in the more chronic forms of nephritis, but may be found in the acute variety as well. This and the granular variety give unmistakable evidence of renal disease.

Granular casts are very friable and vary not a little in appearance. They may be quite long, granular cylinders, or short with irregularly broken ends. Their lateral borders are usually well defined and regular, and the cast seems to be made up of amorphous granules. They may vary in color from very light to a reddish brown. They may also have

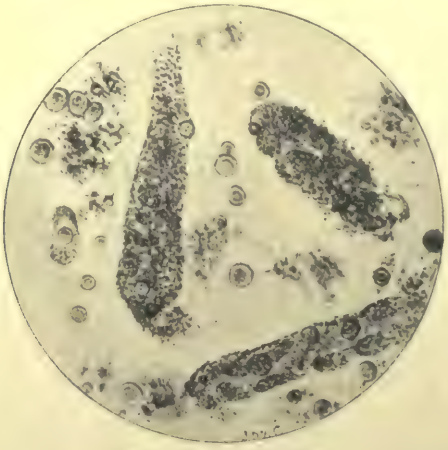
adhering to them scattering red and white blood corpuscles, epithelial cells, and small, highly refracting globules of fat. Granular casts give positive evidence of organic renal disease, and may be observed in the

FIG. 68.

a, epithelial casts; *b*, opaque granular casts (from a case of acute Bright's disease; Roberts).

acute, subacute, and chronic varieties of nephritis. In urine with an unusual abundance of amorphous urates there may appear adherent to a shred of mucus a collection of granular urates, but they will not confuse the experienced observer, and all doubt concerning their nature

FIG. 69.



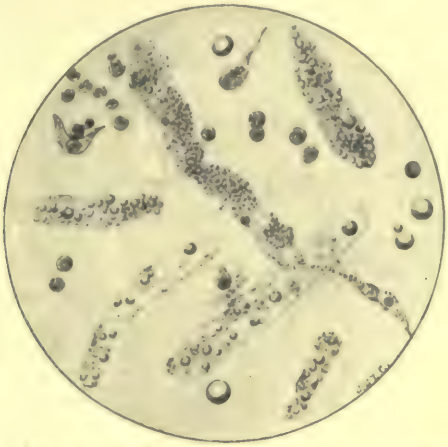
Granular casts (Musser).

may be dispelled by heating the slide, when the urates will quickly disappear.

Fatty casts, granular, and sometimes the more hyaline casts, may

have upon them fat globules and fat and soap crystals of lime and magnesia. Such are called fatty casts. They are found in cases of subacute and chronic nephritis, and are unfavorable in that they indicate fatty degeneration of the renal epithelium.

FIG. 70.



Fatty casts from a case of chronic parenchymatous nephritis.

Hyaline casts are generally shadowy cylinders without color and of various sizes. They may sometimes contain a little granular material, or they may have adhering to them a few corpuscles of both varieties, or an occasional epithelial cell from the tubule. These casts are usually

FIG. 71.

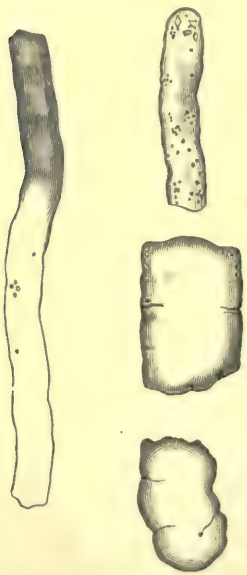
Hyaline casts from a case of acute nephritis: 1, plain hyaline cast; 2, granular deposit hyaline cast; 3, cellular deposit (blood and epithelium).

so faint, because of their hyaline nature, that they may be overlooked; hence it is good practice to stain all specimens when examining for tube casts. They are formed of the albumin which collects in the kidney tubules, and are most abundant in the late stages of chronic nephritis

when the tubules have lost much of their epithelium. They are sometimes found in urine that contains little or no albumin, and when so appearing have been held by some authorities to not indicate disease of the kidneys; but kidneys which are throwing off such casts can scarcely be considered perfectly normal. It is perhaps fair to state that the diagnosis of organic disease of the kidneys when these casts occur sparingly in the urine should be made with caution; and it must be admitted that lesions which sometimes produce them are occasionally transient, and do not leave any trace or unpleasant sequelæ when they disappear from the urine.

Waxy casts are homogeneous cylinders which appear translucent and more solid than the hyaline casts. They may or may not appear segmented, and they may be covered with fat globules, urates, red

FIG. 72.



Different forms of waxy casts (V. Jaksch).

FIG. 73.

Cylindroids from the urine in congested kidneys (V. Jaksch).

and white blood corpuscles, and fungi. They are not pathognomonic of any special lesion, so far as is known, but are found in the various forms of chronic nephritis and in amyloid degeneration of the kidneys. A blue color is produced when these casts are treated with iodine.

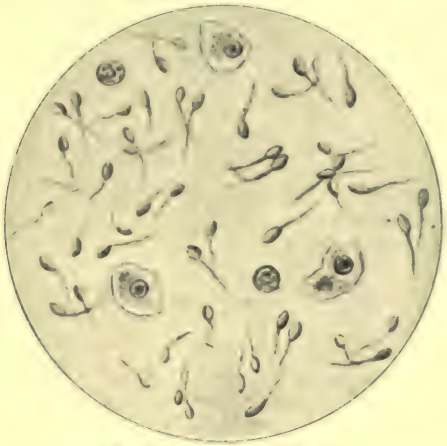
Mucous casts—false casts—are long, ribbon-like cylindroids of mucus. They occur often in the urine of children without other symptoms of disease; also in renal congestion, nephritis, and cystitis.

Spermatozoa may not rarely be found in the urine of male patients

and in that of females after sexual intercourse. They occur in the urine after seminal emissions, whether brought about by sexual intercourse, nocturnal emissions, or masturbation, and in spermatorrhœa. They may also occur after epileptic paroxysms, straining at stool, etc.

Abnormal Growths.—Fragments of tumors have in very rare cases

FIG. 74.



Spermatozoa from urine.

been voided in the urine, but such instances are not common, and when occurring are seldom of such a nature as to furnish much of diagnostic value.

Micro-organisms.—In perfectly healthy urine micro-organisms are not found. When present they may effect entrance into the urine in one of three ways—viz. (1) They may enter from the blood, from the lymphatic channels, or by communication made between some diseased portion of the body and the urinary tract; (2) by introduction into the bladder from without by unclean instruments; (3) by contact with the atmospheric air or unclean utensils after the urine has been voided.

Fission Fungi.—Torulæ cerevisiæ vibriones and swarms of micrococci ureæ quickly develop in urine that is left exposed to the air, and one or more of the fungi operate in the urine to convert urea into carbonate of ammonium, thus changing the reaction from acid to alkaline.

Micrococci ureæ occur in abundance in urine which is undergoing ammoniacal fermentation, and chains of these micrococci appear as strings of beads. In addition various other micro-organisms are often found.

When yeast fungi are present in great abundance sugar will almost invariably be found in the urine. Any of the fungi, micrococci, and pus-producing micro-organisms may be introduced into the bladder by unclean instruments and there set up violent cystitis. In each case the urine will always be alkaline when voided.

In certain acute and chronic diseases pathogenic micro-organisms make their appearance in the urine—*e. g.* in erysipelas when acute nephritis is present; the streptococcus erysipelatis may be found in tuberculosis, especially when it invades the urinary tract; the tubercle

bacillus may be seen in the urine. The tubercle bacillus is much more commonly present in urine than was formerly supposed. It is therefore a good routine practice to examine the urine of tuberculous patients from time to time for bacilli. When they occur in S-shaped masses or curved colonies they indicate tubercular invasion of some portion of the kidney tubule. The method for detecting tubercle bacilli in the urine does not differ in any material way from that employed for their detection in the sputum. Other pathogenic microbes, such as the spirillum of relapsing fever, the typhoid bacillus, the bacillus of glanders, of septicæmia, of ulcerative endocarditis, etc., occur in the urine, and for their detection the reader is referred to special treatises on this subject.

Distoma hæmatobium and its ova are found in the urinary passages of patients in tropical countries. The ova make their appearance in the urine. The sharp angles of the ova irritate the urethra of such patients, so that there is much pain experienced in voiding the urine, and at the end of the process the irritation may be so great that a few drops of blood are passed.

Echinococci hooklets have in rare instances been found in the urine. *Filaria sanguinis hominis* and its ova are sometimes found in the urine of patients who live in the tropics or who have resided in these regions for a time. The worm is filiform in shape, and is about 40 mm. in length. Its ovum is about 0.34 mm. in length and 0.0075 mm. in thickness. The latter are more frequently encountered in the urine. So far as our present knowledge goes, the filaria sanguinis hominis is the chief causal agent in producing the condition of chyluria, which has already been considered (p. 868).

URÆMIA.

By THOMAS D. COLEMAN, M. D.

Uræmia is a diseased condition of the body dependent upon the accumulation and retention in the blood of waste products that should be eliminated by the kidneys, and characterized by such serious functional disturbance of the nerve centres as frequently recurring headache, vertigo, indigestion, flashes of light before the eyes, adventitious sounds in the ears, dyspnœa, convulsions, and coma. In addition, the urine gives evidence of renal disease or is deficient in solids. It is always a secondary or symptomatic disease, and cannot supervene without previous perversion of function on the part of some organ or organs. Such perverted function, whether resulting from abnormal metabolism or from diminished or suppressed activity on the part of the organs of excretion, especially the kidneys, will result in accumulation of excrementitious products in the blood; and these, experimental work and clinical experience teach us, act injuriously on the nerve centres, causing them to send out abnormal nervous impulses that are for the most part purposeless and, not infrequently, disastrous. The accumulation of toxic elements in the blood may be gradual, giving rise to milder and more prolonged nervous symptoms, under which circumstances the organism may, up to a certain limit, accustom itself to the accumulation of the poisonous ingredients. On the other hand, the accumulation of toxic products may be very rapid—as, *e. g.*, in double renal obstruction by calculi—in which case the nervous explosions will be sharper and more violent symptoms will characterize the disorder. We have then two varieties of the disease—viz. the acute and chronic—the symptoms characterizing the two varieties being mainly differences of degree.

Etiology and Pathology.—It was formerly believed that uræmia was invariably the result of a single cause, and from that fact many errors concerning the nature of the disorder have developed. It is now known that the poisons producing the condition are many, and that these may be produced by widely differing causes. While the results obtained are still imperfect, we have brought out here, as strongly as in any diseased condition with which we are acquainted, the value of experimental medicine. Much that we now know concerning the etiology of the disease is due to the accurate scientific work of Bouchard. His own original investigations and his classification of the work of others have given a rational explanation of clinical facts that have long been observed.

Upon reviewing the various theories that have been advanced to ex-

plain the efficient causes of the disorder, one cannot but be surprised at the inadequate explanations that have been accepted by the profession. It is still to be regretted that more positive statements cannot be made regarding some of the subtler processes concerned in its causation. In what follows only those theories that have been generally received will be considered. Among these may be mentioned—

(1) **The Urea Theory.**—For many years it was believed that uræmia was the direct result of an accumulation and retention of urea in the blood—that, the kidneys failing to eliminate this substance, it increased in the blood up to a certain limit, when its toxic properties produced serious perversion of function on the part of the central nervous system. This conclusion was reached because it was observed that there was always a deficiency of urea in the urine of patients who were affected with the disorder, and it may be added that, to the casual student, undue importance was doubtless attributed to the substance because of the supposed import of the word uræmia. The word uræmia does not mean excess of urea in the blood, but is derived from two Greek words, *ουρον* (urine) and *αἷμα* (blood), meaning urine in the blood. In contradiction of this theory it has been shown experimentally and clinically that urea in excess in the blood does not produce the train of symptoms that we are accustomed to regard as uræmic. It has been shown also, in opposition to this theory, that urea may be injected in large quantities into the blood, and not only fail to produce uræmia, but that it is no more toxic than distilled water. Again, it has been shown experimentally that urea is nature's diuretic—that it increases the activity of the cells lining the kidney tubules, just as bile, by moistening the epithelium of portions of the alimentary tract, aids in absorption. We also note clinically that in some cases of hepatic disease where there is diminished formation of urea the urine is lessened in quantity or suppressed. So far from believing urea to be the cause of uræmia, not a few authorities have advocated the subcutaneous injection of urea in the cure of the disease. Still further, it has been observed in cases with double obstruction of the ureters by calculi that death has resulted more quickly than could be accounted for by all the urea that could accumulate in the blood from every source during the time of the attack. In addition, it has been estimated that in order to produce death it requires ten times as much urea as is found in blood of patients dying with uræmia. Lastly, if the normal rate of urea formation is continued and its removal from the blood by the kidneys is entirely suppressed, it has been shown that it would take a man of average weight (of 60 kilos) nineteen days to accumulate enough urea to kill himself. The theory is therefore too barren of confirmation to receive serious acceptance.

(2) **The Hydræmia Theory.**—Traube has advanced the idea that uræmia is nothing more than an accumulation of water in the blood—that it is hydræmia rather than uræmia. He based his theory on the belief that the solids excreted in the urine were constant or fairly so, and that there was a tendency to an increase of water in the blood and tissues. He observed that such patients were frequently dropsical—*i. e.* that there was œdema of the tissues generally and of the brain especially. He therefore concluded that the watery condition of the blood resulted

first from dropsical accumulations, and these in the brain caused anæmia from compression exerted both without and within the ventricles. The theory is untenable, for the following reasons:

(*a*) Many cases of fatal uræmia do not show at the autopsy any œdema of the brain or ventricular dropsy or any cerebral anæmia; on the other hand, cerebral congestion and many small ecchymotic spots may be found. It is a fact, then, that the keystone in the arch of this theory is absent in a great number of cases, and it must fall. If more is desired, it is a fact that uræmic coma supervenes in a large number of cases where the amount of water eliminated is not only well up to the normal, but is even in excess of it. This is well exemplified in cases of contracted and cirrhotic kidneys, in which uræmic symptoms are especially apt to develop, and where the quantity of urine passed may reach as high as 4000 c.c. or more in twenty-four hours.

(*b*) Again, in cases of double renal obstruction from calculi and in animals with both kidneys extirpated death has frequently been found to occur before enough water could have accumulated in the blood to reach any very harmful extent; and in these cases also œdema of the brain has not been found at the autopsy.

(3) **Frerich's ammonæmia theory** is based on his assumption that urea was split up, by ferment action somewhere in the blood, into carbonate of ammonia and water. He based his theory on the odor of ammonia that is frequently present on the breath, in the vomit, and in the sweat of uræmic patients. He observed that urea was present in the urine in diminished quantity in uræmic subjects, and that it would hence accumulate in greater quantity than is found in the blood unless it was split up somewhere in the system into carbonate of ammonia and water. Little of value has been advanced to justify this theory, and it has followed the natural course of all the others that have rested on a purely speculative foundation. In the first place, if Frerich's supposition that carbonate of ammonia is the cause of the line of symptoms that we regard as uræmic were true, is it not fair to infer that patients dying with the disease would have an enormous increase of carbonate of ammonia in the blood? In rebuttal of this, only small quantities of it are found in the blood and mere traces in the urine. Again, urea injected into the blood causes an increased kidney activity, with excess of urea, but no augmentation of the carbonate of ammonia.

(4) **Feltz's and Ritter's Potash Theory.**—These observers contend that uræmic toxæmia is produced by accumulation of mineral substances, chiefly potash, in the blood. This theory is only partly true, since it leaves out of consideration organic substances that will be proved to be concerned in producing the condition.

It has been shown experimentally and confirmed clinically that uric acid cannot be concerned in the production of the disorder—first, because large quantities of it may be injected into the blood without producing anything akin to the disease; secondly, because the normal amount secreted daily is too minute to cause trouble; and finally, because in the gouty there may be large uric acid accumulation in the system without producing uræmia.

It has in like manner been proved that hippuric acid, kreatin, krea-

tinin, leucin, tyrosin, xanthin, hypoxanthin, and guanin are not concerned to any appreciable extent in the production of the disease.

We will next consider a number of ingredients of the blood primarily, and also of the urine, that are not above suspicion, and which are sufficiently important to mention somewhat in detail. It is not now disputed that urine is toxic—*i. e.* it contains substances which produce death when retained in the blood or when injected into an animal. It has been noted, in addition, that the urine of patients suffering with Bright's disease, for example, is less toxic than urine excreted by a healthy man. The logical deduction from this is that the kidneys normally excrete certain waste products that are toxic, and that when the kidneys fail in their function from any cause these toxic products accumulate in the blood and produce the perversion of nervous function that we characterize as uræmia. Now, what are these toxic ingredients?

Bouchard has observed that the kidneys normally secrete in two days and four hours enough of poisonous materials to kill the individual if they are all retained in the blood. Again, it has been shown that the urine excreted at different periods of the day produces different physiological effects when injected into the blood. For example, the urine excreted during the sleeping period is less toxic than that eliminated during the working day, its toxicity being greatest during the first half of the day. Again, the urine of the sleeping period is markedly convulsive, while the urine of the day tends to the production of narcosis. These facts are significant in explaining the physiology of sleep. In like manner, the urine differs in toxicity in diseased states. That excreted in certain diseased states when injected into the blood of another animal will produce convulsions, whereas the urine of other pathological states may be so injected as freely as distilled water without producing them. As has been stated, the urine of Bright's disease is non-toxic, because the toxic principles are retained in the blood. It has also been shown that urine which when fresh kills by producing coma will, when kept for a time, produce death by inducing convulsions, showing that some decomposition of the contained toxic ingredients has occurred.

It has been shown experimentally that the urine contains many toxic principles, which may be separated, and which produce definite physiological effects when injected into the circulation of an animal. From urine evaporated to dryness substances may be obtained which are soluble in alcohol and which produce on injection into an animal somnolence, deep coma, diuresis, and salivation, but do not produce convulsions or a fall in the bodily heat or contraction of the pupils. Other substances may be extracted that are insoluble in alcohol, and which produce physiologically contraction of the pupils, convulsions, and a fall in the bodily temperature. The above excludes aromatic substances, for they would be volatilized by evaporation.

It has been shown, moreover, that urine which has been decolorized by charcoal loses one-third of its toxicity, due to the fact that the coloring matters, one sixteenth of the potash, and all of the alkaloids are removed by the charcoal. These toxic ingredients cause neither convulsions nor contraction of the pupils. The urine has therefore lost these toxic substances by extraction with the charcoal, and, since the coloring matters are thus removed, it is fair to assume that these at least are not

concerned in the production of convulsions. Potash is convulsion-producing, being forty-four times more so than soda. Ammonia as a convulsion-producing agent stands midway between the two. Since one-third of the toxicity of the urine is lost by extraction with the charcoal, and since this contains the alkaloids, it is presumable that at least two-thirds of the toxicity is not due to alkaloids.

Let us now consider the various poisons found in the urine. There are, according to Bouchard, excluding urea, which has been shown to be non-toxic—

(1) An organic principle that is not fixed by carbon, that is soluble in alcohol, and which is narcotic or coma-producing.

(2) There is present in small quantity an organic principle that is not fixed by carbon, but is soluble in alcohol, and, like pilocarpine, produces salivation.

(3) Another organic substance is found that is fixed by carbon and is not a mineral, but is alkaloidal in character. It is insoluble in alcohol, is found in less quantity than the narcotic in the day period, and is convulsion-producing.

(4) A fourth organic substance fixed by carbon is encountered. This element causes contraction of the pupils.

(5) A fifth organic constituent is fixed by carbon. It is not mineral, is insoluble in alcohol, and produces lowering of the body temperature.

(6) Lastly is a fixed inorganic substance that is convulsion-producing, and which is proved to be potash.

There are, therefore, at least six materials found in the blood which are concerned to a greater or less extent in the production of the condition of uræmia. When these are formed at a rate more rapid than normal, it will be seen that unless the kidneys can eliminate them with more than normal rapidity they must accumulate in the blood; and, again, the rate of their production may be normal, but the kidneys through disease or obstruction may not be able to remove them at the normal rate, in which event, like the former, they must accumulate in the blood. It is easy to see that both processes may be concerned in the same case, and the rate of accumulation will determine whether the uræmia shall run an acute or chronic course. It has been estimated that 57 per cent. of the toxicity of the urine is due to the contained mineral ingredients, chiefly potash, and that the balance is due to coloring matters and alkaloids. It is more than probable that some of these under normal conditions neutralize and render others inert.

Do not these results bear out and render intelligible the train of symptoms found in the various uræmic conditions? For example, it has been found that the organic substance which is narcotic acts much more rapidly than the convulsion-producing potash salts, and we know clinically that fatal coma is by far the most frequent termination of uræmia. In like manner, uræmic convulsions are far from infrequent, and according as the former toxic principle or the latter predominates will the phases of the condition be determined. Contracted pupils are sometimes present, more frequently absent. This may be explained by the fact that the myotic toxic ingredient is not found in the urine in large quantity, as a rule, but when in excess causes contraction of the pupils. In the uræmia of cholera more or less contracted pupils have

been observed. Again, a subnormal temperature is frequent, save in the convulsive form of the disease, and salivation has been frequently noted.

The condition, then, may be induced by disassimilation; by certain glandular secretions, such as bile, for example; by articles of diet, especially when mineral substances are in excess, and by intestinal putrefaction; lastly, by faulty elimination on the part of the kidneys. When there is failure on the part of the tissues to assimilate properly, when there is increasing or retained secretion of bile, when improper food, especially that containing excess of potash salts, is used, finally when there is intestinal dyspepsia, uræmia may supervene. When all the above conditions are normal, the disintegration products naturally resulting from cell life and decay will accumulate in the blood if the kidneys fail in their function of elimination. Renal inadequacy may result from acute or passive congestion of the kidneys, producing any of the forms of Bright's disease; obstruction of both ureters by calculi, abnormal growths, or cicatricial bands; retention of urine in the bladder and consequent reabsorption of poisonous products, as may occur in cystitis resulting from atony or paralysis, from enlarged prostate, abnormal growths at neck of bladder; and from stricture and operations on the urethra. Retention in the blood of the toxic products already considered seriously affects the vital condition of the nerve centres, giving rise to many perverted nervous manifestations.

Symptoms.—The symptoms of uræmic toxæmia may be the mild prodromal manifestations of the chronic form or they may be the more violent exhibitions of the acute variety. Chronic uræmia may run a course of many weeks and end in recovery or death. Acute uræmia, on the other hand, may arise from acute obstructive disease of the kidneys, occurring either suddenly or as an exacerbation in the course of the chronic form. In the milder variety the patient may give a history of chronic gastro-intestinal dyspepsia and of a more generous than judicious use of food; or he may have suffered from hepatic disorder and be of a bilious temperament; or he may be the subject of renal disease. He gives a history of pain in the head, chiefly in the occipital region and extending down the neck, or it may be frontal headache or deep pain in the eye-sockets. He may complain in addition of vertigo. There may be irritability of temper, depression of spirits, and a tendency to somnolence. The expression may be vague, pupils contracted, and he may have disturbances of vision with flashes of light before the eyes. Frequently these patients will complain of impairment of sight. This impairment is central, and is brought about by the influence of the toxic substances in the blood on the centre for appreciating visual images. This can be stated with emphasis, because examination of the retinal field with the ophthalmoscope gives no evidence of pathological change in the retina, and the sight returns to the normal within a few days after the exciting cause is removed. The same explanation applies to the metallic tinkling and musical sounds heard in the ears. There may be also tingling of the fingers, and possibly some loss of sensation in the extremities, or there may be painful cramps in the calves of the legs, especially at night. Itching of the skin and a tendency to the development of cutaneous eruptions may occur, and more or less œdema

of the cellular tissue may or may not be present. When dropsy is present, it usually makes its appearance in the loose subcutaneous tissues about the eyes and in the feet, legs, and sometimes in the hands, abdomen, pleural cavity, pericardium, and brain. There is likely to be also gastro-intestinal disturbance, with anorexia, nausea, vomiting, and sometimes diarrhœa. It has been held by some that vomiting and diarrhœa are nature's vicarious efforts at throwing off toxic products that should be eliminated by the kidneys. The respirations will be difficult and noisy, coming on thus chiefly at night and simulating asthmatic attacks. Cheyne-Stokes respiration may make its appearance and last for days or even weeks. The pulse is slow and feeble, reaching as low as 40 beats to the minute, or it may be hard and wiry, reaching from 100 to 140 per minute. There is usually a dusky or sallow, dry skin, but there may be frequently excessive activity on the part of the sweat glands; and it has been noted that these may eliminate urea to some extent, crystals of it having been frequently found deposited on the surface of the skin and hair in cases of pronounced uræmia. This is of value as a diagnostic sign, but since it has been proved that urea *per se* is non-toxic to the organism, the ultimate curative value of such elimination, unless other substances are at the same time thrown off in this way, cannot be great. The urine will on examination be scanty, high colored, and will contain albumin, casts, renal epithelium, and perhaps red and white blood corpuscles, or the quantity may be normal or even enormously increased; the solids will be deficient and the specific gravity low, the color light, and albumin may be absent or present in minute quantity, and casts and renal epithelium will be few and at times absent. This latter type of urine is especially characteristic of general arterio-fibrosis and contracted kidney. When any of the aforementioned symptoms are persistently present without other explainable cause, the urine should be carefully examined.

All the foregoing symptoms may gradually increase in intensity, the headache becoming more severe, the dizziness and depression of spirits and irritability of temper increasing, the somnolence and mental stupor deepening; the sensory disturbances may be augmented on the part of the eyes to greater defects of vision and blindness, and on the part of the ears to greater exaggeration of sounds and deafness: the œdema of the tissues may or may not increase, sometimes becoming extreme and distressing; the respiration may become more and more labored and louder, and the position of orthopnœa must be preserved for even the meagre comfort that the patient can obtain. In addition, the irregular Cheyne-Stokes type of respiration may become more exaggerated: the gastro-intestinal disturbances may advance, there being frequent vomiting, and diarrhœa may be excessive. The urine may become more scanty, with increase in the albumin, casts, and other abnormal ingredients, and even entire suppression of urine may ensue in some cases. On the other hand, the watery portions may continue normal, while the deficiency of solids steadily increases. Finally, the nervous system can bear the strain no longer, and there is a discharge which may or may not be announced by a distinct chill, which is quickly followed by convulsions and coma, or perhaps coma without the development of convulsions.

In acute uræmia many of the foregoing symptoms may be absent, and in the exacerbations of the chronic form it may be that the physician's first contact with the case may be attendance when the active explosions on the part of the nervous system are at their height. In the absence of a reliable history symptoms and signs must guide one. Whether the attack is suddenly ushered in or gradually develops, the succeeding sequence of events is practically the same. As already stated, there may be an initial chill or this may be absent. The chill is quickly followed by convulsions. These may be limited to twitching and tremors of certain groups of muscles, being no different from those of Jacksonian epilepsy, or the convulsions may be general, involving the entire body. The convulsions are epileptiform in character, and when well developed there is marked exophthalmos; the breathing is loud, labored, and high pitched. There are often salivation and frothing at the mouth. The countenance is congested and the pupils usually dilated, but may be contracted, the contraction of the pupils rarely, if ever, being great enough to be confounded with the pinhole pupils characteristic of opium coma. The convulsions may be of short duration, rapidly following each other. Early in this stage there may be periods of consciousness between the convulsions, but as these increase in severity consciousness is lost. The convulsions are usually followed by coma. In this stage the temperature may rise to 106° or 108° F. The convulsions, instead of being followed by coma, may have as a sequel active and violent delirium, in which the patient is so maniacal that he must be restrained, or there may follow instead melancholia with delusions of persecution. While not the rule, these may terminate in hopeless insanity. Death may rarely result without the development of coma. The convulsions so closely simulate those of epilepsy that a history must be obtained and the urine examined before a diagnosis can be made.

In the stage of coma which may develop independently or follow the stage of convulsions there is total loss of consciousness; the countenance is less congested or may be pallid; the pupils are dilated; the breathing is loud, stertorous, or perhaps irregular and of the Cheyne-Stokes type; the pulse wiry and hard, with possibly normal or accelerated rhythm. Vomiting is frequent, and the vomit has an ammoniacal odor due to the presence of carbonate of ammonia, and there may be incontinence of urine and fæces. There may be consequent blindness and deafness, but if the patient survives, these are only transient, both sight and hearing returning after a few days. There may be also following this stage aphasia and hemiplegia. The urine in this stage may be scanty and albuminous, entirely suppressed, or excessive and deficient in solids.

Diagnosis.—The diagnosis of uræmia in its earlier phases is not always possible. When there is persistent occipital or frontal headache without other explainable cause the urine should be carefully examined. If it is found to be albuminous and scanty or deficient in solids, these are probably of uræmic origin. Again, the same urinary anomalies may be attended by gastro-intestinal disorders, torpid liver, and sensory disturbances, in which case the diagnosis is more positive. The convulsions are sometimes of such character that without a history of the other symptoms and an analysis of the urine they cannot be positively diagnosticated from true epilepsy, and where the kidneys are involved

in epilepsy a diagnosis may be impossible. In epilepsy the countenance is usually pale, the intervals between the convulsions are longer as a rule, and in the interval the patient returns to a condition of perfect normality, and there is usually a history of such preceding attacks at intervals, which is not, as a rule, true in uræmic seizures. The urine after epileptic convulsions is usually abundant, light in color, and may be slightly albuminous, but does not contain casts and renal epithelium. In uræmia it is usually scanty, high colored, and contains much albumin, casts, and renal epithelium, with, not infrequently, red and white blood corpuscles. In uræmic convulsions one frequently obtains a history of previous renal disease, and there may be œdema of some part or dropsical accumulations; in epileptic convulsions these are absent. If the convulsions are due to acute renal obstruction, the symptoms will be severe enough to direct attention to these organs, and the suppression of urine will be sufficient to fix the diagnosis. In uræmic convulsions the patient is frequently wildly delirious, even maniacal, and melancholia may follow; in epileptic seizures no such delirium is present, and the convulsion is followed usually by a quiet sleep and a return to his normal condition. In epilepsy the temperature is not elevated and reflex sensibility is not lost. The converse of this is true in uræmic convulsions. In uræmic convulsions there may be an albuminuric retinitis, or blindness and deafness may follow the convulsions. None of these follow epileptic paroxysms.

Diagnosis of Coma.—Uræmic coma may arise suddenly, and it may be, in the emergency, that no history of renal disease is obtainable. It may supervene also in cases where the kidneys are slightly diseased, and, on the other hand, patients may go for a long time without the development of coma when the kidneys are much involved. It has been noted that the system can accommodate itself to the gradual accumulation in the blood of the waste products that should be eliminated by the kidneys, whereas if these are suddenly thrown into the circulation uræmic symptoms quickly supervene. Bartels has explained the sudden onset of coma in dropsical patients by the fact that the dropsical accumulations hold the poisons, and when they become quickly absorbed by the blood uræmic convulsions, coma, and not infrequently death follow. Uræmic coma may be confounded with apoplectic coma, alcoholic coma, opium coma, and diabetic coma, and it is important to review these forms as accurately as possible in making a diagnosis.

Uræmic coma is much less sudden in its onset than that produced by apoplexy or opium-poisoning, and, again, the sequence of events in the uræmic form is much slower than in that produced by opium. In uræmic coma the skin is usually dusky and pallid. If dropsy is present, as is frequently the case, the face may be puffed, especially about the eyelids, and the extremities will pit on pressure. Frequently the abdomen will be found to contain ascitic fluid in greater or less amount. If the intra-abdominal pressure be too great to detect this with accuracy, the trocar or hypodermic needle will settle the matter at once. If dropsy be not present, there will usually be obtained a history of previous renal disease or the acute disorder will direct attention to faulty elimination on the part of the kidneys. The respirations are shallow and irregular, and the expirations are of a higher pitch than those of apoplexy, which are

low and puffing. The breathing may also be stertorous, and finally assume the Cheyne-Stokes character. The pulse is usually irregular and incompressible, having a frequency of a hundred or more to the minute. There may be an aromatic odor to the breath, and the stomach contents, when obtained either by means of the stomach tube or emesis, have an ammoniacal smell. During the earlier stages of the condition the loss of consciousness is not complete, the patient having intervals of consciousness, and then lapsing again into a convulsion or total loss of consciousness. This form of coma may occur in extreme old age, but is not so common as in young and middle-aged subjects. Finally, the urine will give unmistakable evidence of the nature of the trouble. In nearly all the uræmias except the acute obstructive form, when no urine may be obtainable, the urine will be either diminished in quantity or deficient in solids, and diacetic acid, albumin, and tube casts will be found. In some cases also there may be blood, pus, renal epithelium, and bacteria.

In *apoplectic coma* the subject is usually past middle life; the facial expression, if not drawn to one side, is usually more natural than in any of the forms of coma. The complexion is not pallid. The respirations and pulse are slow. Atheromatous arteries are frequently found in these cases. The pupils are almost invariably unequal, and there is conjugate deviation of the eyes. There is usually more or less unilateral anæsthesia, paresis, and finally hemiplegia. The breath and stomach contents have no abnormal odor. The patient cannot be aroused to consciousness, and unless intercurrent renal disease exists the urine will be approximately normal.

In *alcoholic coma* the countenance is flushed and not infrequently bloated. The skin is bathed in sweat. The conjunctivæ are congested and the pupils are not contracted. The respirations are regular and more or less rapid. The pulse is quick, and sometimes small and feeble. The temperature is lowered. There is frequently vomiting, and the breath and vomited matter give forth the odor of alcohol. In the earlier stages the patient may be aroused, but is usually peevish and irritable, not seldom profane in his utterances. His expressions are incomplete, incoherent, and his articulation thick. Unless the kidneys are involved, his urine will be increased in quantity, of low specific gravity, and free from albumin and casts.

In *opium coma*, unless the patient be an opium-eater (and in these cases opium coma is rare) he will usually give a previous history of perfect health. If the drug has been taken by mistake, some trace of it will be found in the patient's surroundings; if through design, this evidence may not seldom be carefully destroyed by the patient. In the first stage the skin is somewhat flushed, and, opium being a diaphoretic and cardiac stimulant, the skin is bathed in sweat and the pulse is strong and full and regular. The pupils are contracted symmetrically to small points. Pinhole pupils are perhaps the strongest link in the chain of evidence. The respirations are slowed, and may reach as low as five or six to the minute, and are obstructive and stertorous in character. The breath frequently gives out the odor of opium, unless morphine or codeine has been taken. The patient can be aroused in the earlier stages, when he will answer intelligently, with good articula-

tion, and in excellent temper, and then quickly lapse again into unconsciousness. As the coma deepens the skin gets livid and clammy, the surface of the body is bathed in sweat, and toward the close of the scene the pupils may dilate, the respirations become shallow and feeble, and the pulse compressible. Unless occurring in a case suffering with renal disease the urine will be fairly normal, except that opium or one of its alkaloids will be found.

In *diabetic coma* the onset is less insidious and sudden, and it is always preceded by the line of symptoms which follow in the wake of diabetes mellitus. Such patients, therefore, are cachectic and emaciated. When the coma is well established the patient is unconscious and completely relaxed; the surface of the body and extremities is cold, the temperature being subnormal, and the pupils, though dilated, respond to light. The breath may sometimes have a chloroform-like odor. Examination of the urine will disclose the presence of sugar, and not infrequently diacetic acid and traces of albumin.

PROGNOSIS.—From the nature of the disease the prognosis is always serious, but not necessarily fatal. Uræmia being the result of the accumulation of poisonous substances in the blood, and since these exert a harmful influence primarily on the central nervous system, and secondarily affect the general nutrition of the body as a whole, the prognosis will depend upon the ease and rapidity with which these toxic principles may be eliminated from the system by all the channels of excretion. In acute uræmia due to obstruction of the ureters by calculi the prognosis will hang on the patient's vital condition and the skill and celerity with which the obstructing bodies are removed, so that the kidneys may again resume their function of elimination. Acute uræmia when induced by acute nephritis frequently develops in the robust, and if the treatment is vigorous and instituted early enough, the prognosis is not necessarily unfavorable. A return to the normal may be complete or the condition may exist long enough for permanent renal disease to follow as a sequel. Acute uræmia developing in the course of any fever, as yellow fever, scarlet fever, or in diphtheria, is an unfavorable complication, and the prognosis will depend on the patient's general nutrition and his power of reaction to remedies directed to eliminating the poisons from his system. If taken in the early stages and the patient's physical condition is fair, the prognosis need not be bad. In cholera there is frequently anuria, and, though more than the normal quantity of water may be lost in the copious diarrhœal discharges, and with the water possibly some of the toxins (though this is yet unproved), it is still a fact that both the bowels and skin together cannot eliminate enough of these to avert fatal uræmia, so that in these cases the prognosis is almost invariably bad. In puerperal eclampsia the prognosis will depend, first, on whether medicines which favor elimination of the poisons retained in the blood can act with sufficient rapidity to remove such products before the system is overwhelmed, and, second, on quickly terminating the labor by delivering the fœtus.

While statistical tables are wanting, and while individual cases may occasionally stand out in strong contrast, I believe that the prognosis for the ultimate favorable outcome is better in acute than in chronic uræmia. It is true that the poisons may accumulate with great rapidity

and thus overwhelm the nervous system, and oftentimes before the organs of excretion can be made to act, but it is also true that such cases are more frequently in better physical condition, and therefore better able to respond to stimulation, than are chronic uræmics. The organs which most seriously affect the prognosis are the kidneys. If these organs are so much diseased that true urinary secretion cannot take place in sufficient quantity to relieve the blood of its weight of poisons, the prognosis will be bad.

In chronic uræmia, on the other hand, the poisons, though accumulating slowly, are none the less surely affecting the integrity of the nerve centres and interfering with the oxygenation and general nutrition of the tissues, so that, though the battle may be long and weary, characterized by sharp encounters, and, it may be, closing with a siege, it is in the end a losing one, and the organism must finally surrender. For the reasons already given uræmic convulsions or coma, or both, occurring in the course of chronic uræmia must be regarded with more serious concern than those brought on by the acute disorder. I have said that uræmia may result from retention of urine in the bladder and consequent absorption of the toxic products by the blood, whether such retention be produced by atony of the bladder, by obstructive growth in the neck of the bladder, or by operations on the urethra. It is easy to see that if proper operative procedures are instituted and suitable drainage and irrigation of the bladder are effected, the immediate symptoms may not only be relieved, but the patient may be permanently cured. In such an event the prognosis will depend on the general condition of the patient and the skill of the operator.

Treatment.—There are few diseases that require greater care and skill in their treatment than uræmia. Since it has been proven that uræmia is the result of the action of several poisons on the central nervous system, the treatment is more complicated, and still, on the whole, more scientific.

Hygienic Treatment.—Elimination of the poisons by every emunctory of the body, and their reduction in the blood to a minimum by regulating the diet, should be effected. To this end the food taken should be regulated rigidly by the physician, and he should see to it that digestion is properly carried out and that the skin, bowels, and kidneys shall act freely. While it is true that some poisons are eliminated in small quantities by the skin and bowels, it is also a fact that these two channels of elimination in this disorder are not regarded with such importance as formerly. It has been shown that urea has been eliminated in small quantity by the skin, and in still less amount, if any, by the bowels; and it would be interesting to know if any of the efficient causes of uræmia may be eliminated to any very considerable extent by these two channels. It has been shown that uræmia results from the accumulation in the blood of poisons that should be excreted by the kidneys. This accumulation can result from three causes—viz. from the food taken, from cell metabolism, and from inadequacy of the organs of excretion, chiefly the kidneys. These causes must therefore be considered in outlining the treatment. So, then, in all cases the diet must be restricted to food that is scanty in mineral salts, especially potash—that is easy of digestion and does not encourage putrefactive processes in the intes-

tines; and cell activity must be reduced to the lowest limits compatible with healthy function. To this end excessive and exacting physical and mental exercise must be prohibited, and the patient must be made to live out of doors as much as possible, for oxygen in generous quantity neutralizes some of the harmful effects of certain of the poisons already considered. Lastly, in order to purge the blood of its abnormal supply of poisons, the kidneys, skin, and bowels must be stimulated to proper activity by every available means.

Again, it has been shown that according as the poisons rapidly or gradually increase will the type of the disease be acute or chronic. The treatment then resolves itself into two classes—that of the chronic and acute types of the disorder.

Hygienic Treatment of Chronic Uræmia.—In this condition the diet should be rigidly restricted both in quality and quantity. The food should be limited to articles of diet that are easily digested. Milk is, beyond question, the best of all foods in this disorder, and severe cases should be limited to it exclusively. The points of advantage are—it is easy of digestion, it contains a minimum amount of potassium salts, it is diuretic, and represents a concentrated form of nourishment. In addition to milk, eggs may be allowed prepared in any way that is agreeable to the patient. Cooked starches may be allowed in the form of bread that is not sodden; oatmeal or cracked wheat with or without sugar and cream; beans, peas, and potatoes well stewed or baked. Thoroughly cooked green vegetables and fruit that is preferably stewed may also be allowed. Meat should be entirely prohibited or allowed in quantity not more than one quarter to one half pound two or three times a week. When it is allowed it should be thoroughly cooked, as in this way many of the contained putrefactive elements may be destroyed. It may be necessary to look further to the digestion by the administration of medicinal aids, but this will be considered under the medicinal treatment. The individual case may also make it necessary for the physician to eliminate any or all of the above articles save milk. A diet limited exclusively to milk has for a long time been used, and sometimes with gratifying results, in the treatment of chronic nephritis. In addition to the care of the diet, other hygienic rules must be followed. The bowels should be regulated, and two or three liquid stools should be passed each day. The skin should be made to act up to its fullest power of elimination, and to that end a hot bath should be taken each day, after which alcohol may be applied, and general friction employed either with a crash towel, leather glove, or flesh brush. All exacting physical and mental exertion must be prohibited, for it has been shown that the urine of one who has exercised violently or who has subjected himself to unusual mental tax and worry is much more toxic than normal urine; so that the patient's usual vocation should be left off, and only such exercise as promotes normal physiological work on the part of the tissues, and light, entertaining literature and pleasure, should be allowed. An abundant supply of oxygen is beneficial, as this in the system tends to destroy certain of these toxic products, so that it is well for the patient to protect himself from sudden chilling of the surface of the body by appropriate clothing, and to indulge in out-of-door recreation that does not require much physical effort or mental concentration.

The inhalation of pure oxygen is recommended by some as being beneficial in these cases.

Medicinal Treatment.—The medicinal treatment should also be directed to improving the assimilation, maintaining the strength of the patient as far as possible, and the elimination of the various toxic ingredients by way of the three emunctories. To this end the digestion must be watched, and it may be necessary to administer pepsin in some of its forms. In flatulent dyspepsia, that is so common in these cases, I have found a pill composed of scale pepsin 5 gr., salol 5 gr., and powdered cinnamon or oil of black pepper 1 gr. especially useful. The pepsin aids in gastric digestion; the salol, splitting up in the intestines into salicylic acid and phenol, together with the cinnamon or other spice, are efficient in preventing putrefactive fermentation that has been proved to be deleterious. One to two of these pills may be administered after meals. As additional intestinal antiseptics salicylate of bismuth and salicylate of magnesia, benzoate of soda in 5 gr. doses, may be mentioned as useful. Among these, benzoate of soda in doses of 5 gr. every two hours is most highly regarded by some. One to two drachms may be given each day if the case requires it. Bouchard has directed attention to the fact that charcoal will extract some toxic products from urine, and that when administered by mouth the urine is in like manner rendered less toxic. It reduces fermentation in the intestines, and in drachm doses four times a day or oftener gives satisfactory results in chronic uræmia, especially when intestinal indigestion is marked. I do not accept the position taken by some authors that purgatives and diaphoretics are of little use. Granted that they do remove much water and very few toxic ingredients, leaving the remaining blood more concentrated—more full proportionately of toxic materials. The quota of water is soon supplied by that taken in the drink, and if the diaphoresis and purgation have removed only a small quantity of the poisonous elements, it still is a point gained, and practical experience teaches that the gain is not inconsiderable at times. It has been shown beyond question, and is now generally accepted by authorities, that nearly 50 per cent. of the toxic elements in the blood of uræmic cases is represented by the salts of potassium; hence these medicinal salts, whether administered in the form of purgatives (as cream of tartar), diuretics (*e. g.* acetate of potassium), or as sedatives (*e. g.* bromide of potash), should be absolutely interdicted.

Calomel alone or in combination with other purgatives is excellent. It may be given in doses ranging from gr. $\frac{1}{4}$ to gr. 1 at bedtime or in larger amount, and oftener if the case is urgent: $\frac{1}{10}$ gr. doses of calomel repeated until six or eight are taken, and given at such intervals as the individual case may require, are most beneficial. In this dose it plays a quadruple rôle: it acts mildly on the liver, increasing its activity and favoring the elimination of bile. It will be remembered that defective metabolism on the part of the liver leads up to uræmia. In addition, calomel is to a certain extent antiseptic. It is a laxative in these doses, and finally it is diuretic. Among the other agents that act upon this channel of elimination may be mentioned sulphate of magnesia, in 2 to 4 drachm doses; effervescing citrate of magnesia in 8 to 16 ounce doses, and repeated every two hours if necessary to produce watery stools.

Elaterium is a drastic purgative, producing large watery stools and acting very quickly. This may be given by the mouth in solution in a little whiskey or brandy or hypodermically in doses of gr. $\frac{1}{4}$. If elaterin be used instead of elaterium, the dose should be gr. $\frac{1}{16}$. It is well to bear in mind that in all cases where the dropsy is general drugs given by the mouth or rectum will be more efficacious than if they are administered hypodermically, for the reason that the blood does not readily take up medicinal agents from tissue that is infiltrated and œdematous. Other purgatives, such as rhubarb, jalap, and scammony, may also be employed. To induce increased functional activity on the part of the skin both the dry and moist hot packs may be mentioned as efficacious. These are not usually employed except when there is urgent demand to avert an impending convulsion or coma.

Diuretics as a class have been objected to on the ground that they irritate the already embarrassed kidneys. From the almost universal testimony of recent authorities on this subject it would seem that this objection is theoretical rather than practical. If clinical experience teaches anything, it is that our greatest promise of success in these cases lies in the judicious employment of diuretics. It, however, goes without saying that for any diuretic to be effective there must remain some normal kidney tissue to respond to the stimulating influence of the diuretic.

Milk is a natural diuretic and its effect is wholesome. Chief among the medicinal diuretics may be mentioned digitalis. It is non-irritating to the kidneys, increases the heart's action, and augments the intra-renal arterial and capillary pressure. By the increase of pressure it overcomes the renal capillary obstruction or stasis and thereby augments the flow of urine. The preparation of this drug that gives the best results is the infusion. This should be freshly made, and of it a tablespoonful three times daily may be given, or every three hours if the demand is urgent. The latter interval may be often required, for very frequently the kidneys, so great is their involvement, are slow in responding to stimulation of every kind.

Again, when digitalis proves irritating to the stomach, as it frequently does, strophanthus may be used with almost equally good effect, and Dujardin-Beaumetz claims that its action is more immediate. He states, without qualification, that he has never seen the drug produce nephritis, as some have claimed. Sulphate of sparteine may also be employed, but its action is less reliable than either of the two preceding. Citrate of caffeine is also highly commended by many authorities. It may be administered alone, or better with benzoate of soda in the proportion of 2 grains of caffeine and 5 of benzoate of soda every four hours, and oftener if necessary. These may be used either by the mouth, rectum, or hypodermically.

Germain See and Dujardin-Beaumetz, noting that diabetic patients were polyuric, have experimented with lactose and glucose. Their results experimentally and clinically were such as to lead them to suggest these remedies in the oliguria of uræmia. Very large doses of them are given, as much as 100 grains dissolved in 1 to 2 litres of water being administered daily. Four to six effervescing tablets of carbonate of lithium may also be used with beneficial effect when given in connection with one or more of the above diuretics.

When the fluid accumulates in the abdominal cavity to considerable extent, the resulting discomfort may be so great that a more rapid removal of the fluid than the ordinary channels of excretion will give is required. A ready and quick method will be its removal by means of the trocar. If the fluid contains even a part of the poisons that have been attributed to it, how greatly must the rapid removal relieve the already overburdened system! Again, the œdema in the legs is at times so great that the circulation becomes impaired and there is danger of extensive sloughing of the skin. In such cases acupuncture will frequently result in the removal of sufficient fluid to relieve the tension until the kidneys can be made to act.

At times the heart may be so weak that it requires to be sustained by the use of strychnine, whiskey, or brandy and Hoffman's anodyne. The combination of whiskey and Hoffman's anodyne is at times very efficient, and especially grateful to the patient.

To counteract the nervous symptoms of headache and insomnia and irritability, bromide of soda may be administered in doses of 30 grains once, twice, or three times a day, and oftener if necessary. This should be employed in connection with the drugs which favor elimination on the part of the kidneys, skin, and bowels. McKenzie recommends cannabis indica in doses of gr. $\frac{1}{12}$ to gr. 1, night and morning, for the cure of uræmic headaches. When the case is well advanced and the above-mentioned symptoms, with great distress of breathing, come on, as they frequently do, at night, I do not know any drug that gives the relief of $\frac{1}{4}$ grain of morphine at bedtime, and oftener when the symptoms are more urgent. Not only is the patient made more comfortable, but in my opinion a convulsion may often be averted by its prompt use.

Treatment of the Acute Stage.—Though convulsions sometimes come on without warning, there are frequently premonitions of its advent and it is then that prompt action is demanded. V. Jaksch mentions prominently among these the appearance of diacetic acid in the urine. In this stage hygienic treatment is out of the question, and only those drugs which act energetically and powerfully must be employed. To this end free purgation must be produced, and in this elaterium is applicable because of its rapid action. The hot pack may also be used to induce sweating. Better than this in some cases is the use of pilocarpine in its effect on gland secretion. To obtain the full physiological effect of profuse sweating, ptyalism, and increased kidney activity, it must be administered either by mouth or hypodermically in doses ranging from gr. $\frac{1}{16}$ to gr. $\frac{1}{3}$. The great drawback to the use of this drug lies in its very depressing effect on the heart, so that its use in asthenic cases is contraindicated. It is best to administer strychnine and whiskey whenever the drug is administered to patients who are not robust or those who have disabled hearts. Veratrum viride in sthenic cases acts well, and is especially efficacious when given with morphine. It is recommended especially in puerperal eclampsia, but has the same disadvantage as the preceding drug. It is a great depressant, and cannot be safely employed in asthenic cases. It may be given hypodermically in doses of 5–10 minims, and may be repeated until the arterial tension is near the normal.

Dry and wet cups and hot fomentations over the kidneys should

also be resorted to in order to bring about activity on the part of these organs.

When the convulsion is well established it is imperative to diminish the nervous discharges and terminate the paroxysm as quickly as possible, else death may ensue. For this purpose the administration of chloroform by inhalation has been recommended. As Loomis states, "its only clinical effect is to control muscular spasm, and in a large proportion of cases it fails to give more than temporary relief to those patients who pass from successive convulsions into complete coma, and die without any apparent neutralizing effect from the chloroform;" and he claims that it is prejudicial to ultimate recovery. I may add that it produces the additional disadvantage of inducing renal congestion, and this to the kidneys already overtaxed is far from advantageous; but the condition can be so urgent that its employment may be sanctioned until the effect of other remedies of greater curative value can be felt.

Bromide of potash should not be considered for reasons already given. Bromide of soda and chloral are both good, but their action is too slow.

Kinnicutt and Peabody recommend the use of urethane. They recommend the administration of from 6–7 drachms in twenty-four hours. They claim that it is less irritating and less depressing to the heart than is chloral. The drug may be administered hypodermically or by enema.

In plethoric subjects suffering with an acute attack of uræmia no remedy is so quick, so efficient, and so rational as venesection. It removes from the system a large amount of poisons in the blood, and as much as from one to two pints may be withdrawn. When this is done it is well to inject normal salt solution either into the blood stream or subcutaneously by means of Allen's surgical pump, or into the rectum. Bloodletting in plethoric subjects is most efficient, but in those suffering with chronic uræmia it is at best a makeshift, and many patients are so weak from exhaustion that they are not able to stand the depletion.

The drug which gives, perhaps, the most satisfactory results in the convulsions of uræmia is morphine, administered hypodermically, in doses ranging from gr. $\frac{1}{4}$ to gr. $\frac{1}{2}$, and repeated every two hours if necessary to control the convulsions. Loomis has given as the advantages possessed by morphine—

First, that morphine can be given hypodermically to some if not to all patients with acute uræmia without endangering life.

Second, that the almost uniform effect of morphine so administered is, first, to arrest muscular spasm, and, secondly, to establish profuse diaphoresis.

The drug surely does not interfere with elimination, is in nowise irritating to the kidneys, and in my experience has given all the gratifying results that he has enumerated.

Treatment of the State of Coma.—In this stage all the drugs that we have found to be of service in the chronic stage must be pushed to their physiological limit. To this end purgatives must be used to produce watery stools—infusion of digitalis in $\frac{1}{2}$-ounce doses every three hours to increase the secretion of the kidneys, and Bouchard has suggested

the high rectal injection of cold water to constringe the vessels in the abdominal cavity, and thus increase the tension in the renal arteries and capillaries.

Dry cups and hot fomentations over kidneys are frequently of service and should be applied. Finally, in sthenic cases pilocarpine or blood-letting may be employed.

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